ANNALS OF SURGERY

VOL. 127 APRIL, 1948 No. 4

MARROW-NAILING OF RECENT FRACTURES, PSEUDARTHROSIS AND BONE PLASTIC

EXPERIENCES IN 100 CASES

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THE MARROW-NAILING by treatment of fractures of the long bones has as its originator Küntscher, of Kiel, Germany. In 1940, he published[1] his first results with the new method. I have used marrow-nailing in certain cases since 1943, and have, in Acta Chirurgica Scandinavica, vol. **90**, 89–104. 1944, described our first 28 cases. Since that time we have undertaken marrow-nailing in 72 additional cases—and we are now rather experienced in this field. Our results are still encouraging. On account of the war and the interrupted communications between the various countries, the method of medullary-nailing has not become as well known as it deserves. To judge from the scientific American literature which has come to Sweden during the past few years, the method does not seem to have been used in America, and I have, therefore, thought that a report of our experiences might be of interest to American surgeons.

The aim of medullary-nailing is to keep the bone fragments in position by means of a long nail inserted into the marrow cavity. As the nail is driven in from a hole chiseled in the corticalis far from the site of the fracture, the latter is not exposed and it is not a question of open reduction. The nail is U- or rather V-shaped and does not fill the whole marrow cavity and, thereby, causes very little damage to the bone marrow and the endosteum but gives, nevertheless, a firm fixation. The nail reaches, as indicated in Figure 1, the endosteum only in three narrow places, and as it is comparatively thin (1.5–2 mm.) it only causes a moderate compression of the marrow. Through its angulation and the hardness of the steel, the nail, in spite of its thinness, is very resistant to flexion and if it fits well into the marrow cavity it gives a very firm fixation.

In fractures of the femur the nail is inserted from the upper surface of the trochanter major, either percutaneously or through a small incision. No chiseling is needed as the bone here is very soft. When inserting the nail into the other long bones, such as the humerus, radius, ulna or tibia, a small hole must be chiseled or bored through the corticalis at a suitable distance from the site of the fracture.

FIG. 1.—(a) Medullary Nails. (b) (Below) showing detail of design and cross-section of nail *in situ*.

a

FIG. 1b

The difficulty is to get such a good closed reposition that the nail can be inserted from one fragment directly into the marrow cavity of the other. Several apparatuses for facilitating this reposition have been constructed. Küntscher attaches great importance to the closed reposition, as the exposure of the fracture site and the open reposition involves risk of infection and outer complications. Nowadays, with penicillin, this risk is, however, very slight. As my cases show, it is sometimes necessary to use open reposition. If the closed reposition is successful the marrow-nailing is only a small operation, which causes the patient only little strain and little risk. Here, we have one of the great advantages of the new method over the ordinary osteosynthesis.

The nail must be inserted under fluoroscopic supervision, or repeatedly checked by several roentgenograms. We have used the latter method and found it satisfactory. One may also, in order not to risk having to take out the nail and change its position, first insert a "leader" (Kirschner needle), which is done in operations on fractures of the femoral neck.

We used nails manufactured by Ericsson's Instrument Company, in

Gothenburg, Sweden.* These are made of rustless steel. Our first nails, however, were not sufficiently strong and were bent at the site of insertion or later through the strain of the fracture. The new ones are more satisfactory. The nail must not, however, be manufactured of too hard steel, which would prevent the flexion necessary to insert the nail into the marrow cavity from the small hole in the corticalis. The instruments needed are very simple (Fig. 2). Of course, it is necessary to have a good apparatus to withdraw the nail if it happens to lie in a wrong position and after healing of the fracture. It is, naturally, necessary to have several nails of different lengths and widths at hand (from 10 to 45 cm. [Fig. 1]).

Up until January 5, 1946, we have used marrow-nailing in 100 cases of which 69 have been recent fractures, 24 pseudarthrosis and 7 cases of bone plastics. The cases are divided as seen in Table I.

TABLE I

	Recent Fractures	Pseud-arthrosis	Bone Plastic	Totals
Femur	13	6	3	22
Tibia	28	3	1	32
Humerus	15	13	3	31
Radius or ulna	12	1	..	13
Metacarpal	1	1	..	2
	69	24	7	100

As it would require too much time to describe all our nailed cases, a brief account, only, will be given of some characteristic cases in each group.

A. RECENT FRACTURES

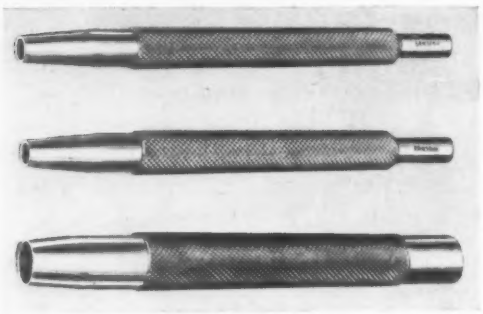
Case 1.—Man, age 31, with an oblique fracture of the femur. When extension treatment did not result in good position and roentgenograms showed muscle interposition, medullary-nailing was done on April 30, 1943. The interposition made open reduction necessary. The nail was inserted from the upper surface of the trochanter major. A good position, with firm fixation and complete freedom from pain was immediately obtained. The patient could sit up after a week, and three weeks later he was able to bear weight on his leg. When he was discharged, seven weeks after the operation, he walked easily with a cane. There is an early and powerful callus formation. The nail was later removed.

Case 2.—A woman, age 45, sustained a complicated transverse fracture of the right femur on November 14, 1943. Immediately upon admission operation was performed, with careful removal of all the injured tissues and open reposition of the fragments. A marrow nail was inserted from the trochanter major. Primary suturing and healing. As the fixation was not absolutely stable, another nail was inserted a week later, with better results. The patient got up soon, and was able to bear weight on her leg four weeks after the second operation (Fig. 3). The nail was removed after six months.

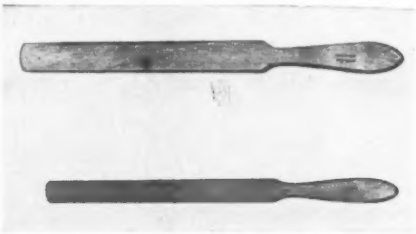
* Zimmer Manufacturing Co., Warzawa, Indiana.

Case 3.—Man, age 33. Splinter fracture of the humerus, with bone interposition. Extension treatment did not result in good position. Open reduction and marrow-nailing. Firm fixation was obtained. No plaster splint was applied. Six weeks after marrow-nailing shows roentgenographic consolidation of the fracture (Fig. 4).

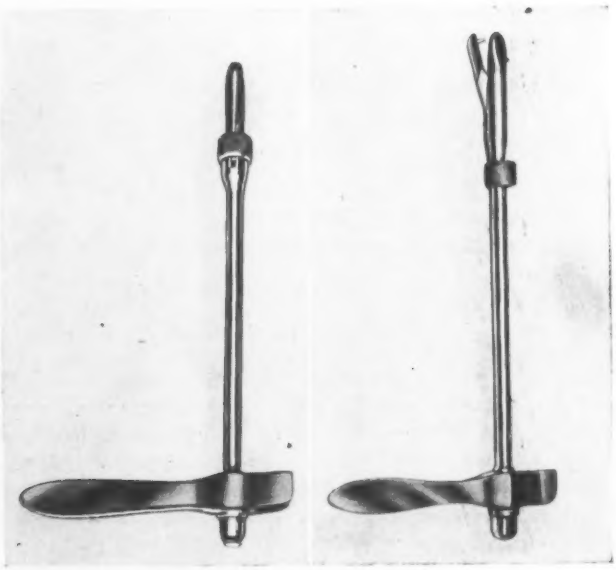
Case 4.—Man, age 60. Fracture of the lower leg. Extension treatment did not result in good position. Instead of osteosynthesis we chose medullary-nailing. The nail



Driver.



Striking Tool.



Nail-drawing instrument.

FIG. 2.—Instruments used in marrow-nailing.

was inserted through a small hole chiseled out in the upper part of the tibia on February 8, 1945. Good position was obtained, but when the distal stump was too short to give as firm fixation as was desired a plaster encasement was applied for some weeks. The leg could bear weight six weeks after nailing (Fig. 5).

Case 5.—Man, age 20. Transverse fracture of the radius and ulna. After repeated reductions a good position of the ulna was obtained, but not of the radius fragments.

Medullary-nailing was, therefore, chosen instead of osteosynthesis, the nail being inserted through a small hole in the distal end of the radius. A plaster splint was applied for four weeks. The man had recovered his working capacity six weeks after operation.

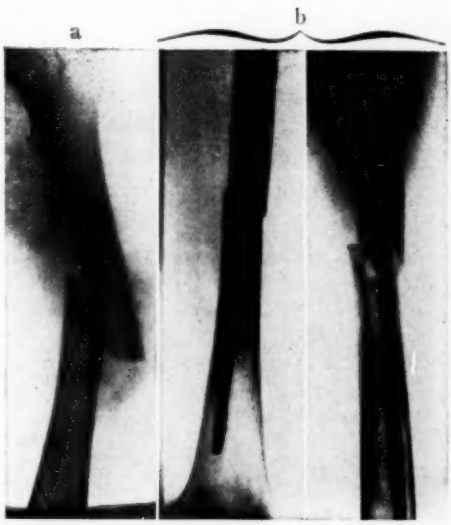


FIG. 3.—Case 2: (a) Open fracture of the femur. (b) After marrow-nailing.

It is extremely important to the healing of the fracture that absolutely firm fixation is obtained in medullary-nailing. For this, one must choose a nail which fits well into the marrow cavity and ascertain that the latter is of fairly even width. This is the case in the cavity of the femur and to a certain degree in the radius and ulna, but the tibia and humerus often have rather symmetrical marrow cavities. Thus, the best results are obtained in nailing of femoral fractures. The nail must be wide enough so that it establishes firm contact with the wall of the marrow cavity, mainly, in order to prevent rotation between the fragments. One can estimate from the size of the marrow cavity in the roentgenogram about what size nail to choose. According to Fischer, with a focal distance of 75 cm. the marrow cavity is

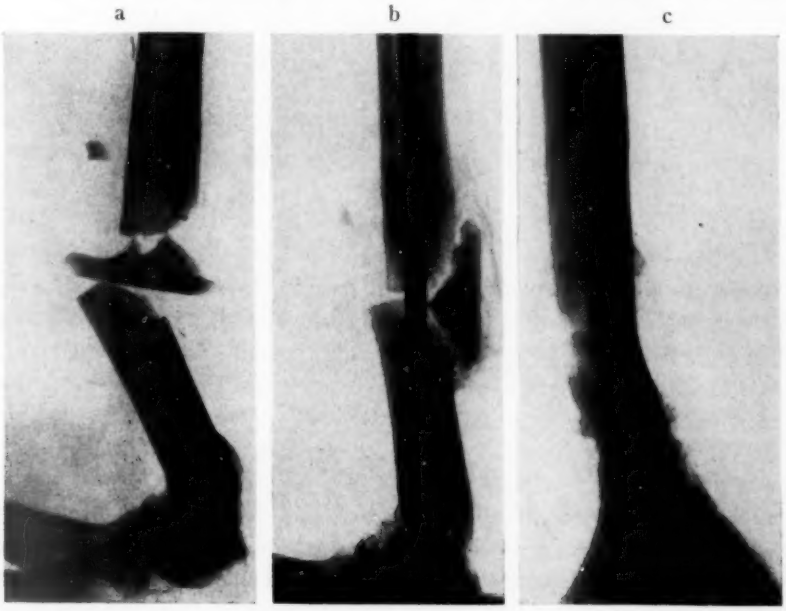


FIG. 4.—Case 3: (a) Fracture of the humerus, with bone interposition. (b) Three weeks after operation. (c) Bone consolidation six weeks after marrow-nailing.

really 1 mm. smaller than it appears on the roentgenogram. The conditions are not so favorable in the tibia as they are in the femur, for, here, the cavity is narrowest in the middle. Consequently, it is easy to obtain firm fixation of fractures situated in about the middle of the tibia but not of ones situated more distally or proximally.

If full stability is not obtained with one nail, it is wise to insert another one (Case 2). This needs to be done more often in the cases of bones with uneven medullary cavities (tibia and humerus) than of ones with an even canal.

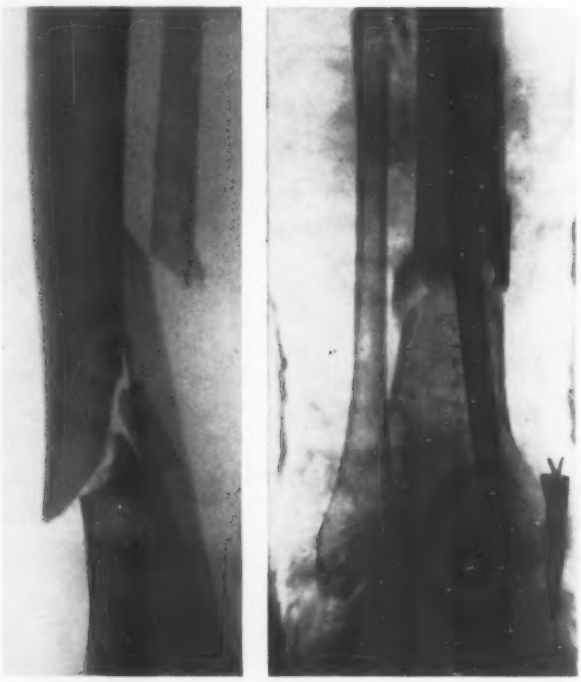


FIG. 5.—Fracture of the lower leg before and after marrow-nailing.

Medullary-nailing has given the best results in fractures of the femur. The advantages of this method are also greatest in these cases. Previously, the patients had to lie in bed for two or three months and undergo extension treatment for long intervals, a procedure which is irksome to the patient and requires much attention from the hospital staff. The long stay in bed also involves the risk of complications in the air passages, at least with older persons. Now these patients can be allowed up early, and can soon begin to bear weight on their leg, thus avoiding stiffness in the joints and muscular atrophy. Another great advantage is that the patients are immediately rid of pain. It is also maintained by Küntscher, and others, that the nailing accelerates the formation of callus, thus, considerably reducing the time before the patient is able to resume his normal occupation.

Fischer also recommends medullary-nailing for supracondylar femoral fractures. The greatest advantage in these cases is that the nail keeps the fragments in position, which is difficult to obtain with extension treatment. When the nail can only be inserted 3–4 cm. in the distal fragment it is necessary to apply a plaster encasement for some weeks, and early weight-bearing is out of the question.

The *advantages of medullary-nailing,* particularly in femoral fractures, are, in my experience, and that of others:

1. Shortened stay in bed.
2. Simplified after-treatment—no extension.
3. Reduced pains and other subjective troubles.
4. Less risk of stiff joints, muscular atrophy and circulatory disorder.
5. Shorter hospitalization and probably earlier acquirement of working capacity.

No definite agreement has been reached about the *indications for medullary-nailing.* Some surgeons have narrow indications, and others very wide ones. Küntscher says that the method is suitable for all transverse, oblique and spiral fractures of the long bones, and Pascher[2] goes so far that he considers it absolutely indicated in all transverse, all oblique fractures with a poor healing tendency or where there is risk of slipping, and in all fractures in old persons, where long stay in bed should be avoided. Böhler, who is a strong opponent of open treatment for fractures, recommends the method warmly for gunshot fractures, thus, open fractures, and seems to be a strong advocate of medullary-nailing, on the whole. Others, for example K. H. Bauer,[3] reserve the method for more special cases, *e. g.,* for times when it is necessary for some reason to make an open reduction.

The marrow-nailing involves, of course, certain risks. Above everything, we must think of the danger of bone *marrow destruction, fat embolism* and *osteomyelitis.* Experience has already shown that the damage which the nail causes to the bone marrow is of no or little practical significance. It is recommended, however, that the nail be removed when full consolidation is established. We have done that in the majority of our cases. The removing of the nail is a simple operation. After a few months the nail generally lies fairly loose in the marrow cavity. On the other hand, there is a certain risk of fat embolism. At least two deaths from fat embolism after medullary-nailing are reported (Küntscher and Häbler). Detailed information is lacking, and it is impossible to decide whether the fat embolism occurred as a result of the fracture or of the nailing. Osteitis has developed in a few cases (Küntscher, Fisher, and others.) In most of them it was a question of complicated fractures. Extended osteomyelitis, generally, does not develop, but only restricted osteitis, with local sequestration, mainly due to the fact that the pus in the marrow cavity is led off along the nail. Thus, according to Küntscher, Böhler, and others, there is never any enclosure of pus in the cavity with rising pressure, which is generally considered to be the cause of extending osteomyelitis. Küntscher says that whenever there is infection,

one should drain, so that the pus is easily able to drain off, but not remove the nail since it generally heals in spite of the infection. That is also my experience. If the nail is removed, the infection grows worse because the fragments no longer lie still.

B. PSEUDARTHROSIS

The treatment of pseudarthrosis has always caused surgeons great difficulty. Well-nigh countless methods have been tried but none has so far proved itself to be supreme. The problem has now been actualized through the increase of the number of cases due to the gunshot injuries sustained during the war. Even here, medullary-nailing has been used and I have, up to date, nailed 24 cases of pseudarthrosis. The results are thus far satisfactory. In the cases which I have been able to follow for a sufficiently long time, the bone-healing was satisfactory except in three cases where inflammation recurred after operation.

My experience is that the marrow-nailing will simplify the pseudarthrosis problem considerably. The literature is still very scanty. K. H. Bauer, who otherwise is reserved concerning this method, recommends it in cases of pseudarthrosis, and so do Böhler[4] and Cellarius,[5] from Kirschner's Clinic, who report the results of 18 cases which were earlier treated in vain by other methods. In at least 15 of these cases bone-healing took place within 6–8 months after marrow-nailing.

In order to hasten the bone-healing, the marrow-nailing may be combined with other operations, such as bone transplantation, either in the form of bone clips, according to Levander, or by covering the pseudarthrosis with a larger bone piece. Often, marrow-nailing alone is sufficient. All my cases, with the exception of four, were Finnish soldiers injured in the war. Appended are reports of some characteristic cases:

A. PSEUDARTHROSIS OF THE FEMUR

Case 6.—A 22-year-old sergeant sustained a gunshot fracture of the femur a few fingersbreadth below the trochanter minor, on October 22, 1941. He was treated in Finland with extension, plaster encasement, *etc.* He arrived at Sahlgrenska Hospital, July 22, 1942, with his leg in a plaster encasement. When the plaster encasement was removed, the bone ends slipped apart. The wound was revised, sequestrotomy performed, and wire extension instituted. No healing took place, osteosynthesis with transplantation of bone clips, according to Levander, was done in December. Consolidation did not result, and, in March, 1943, medullary-nailing was done after revision with freshening of the bony ends. Firm fixation was obtained at the site of the fracture; and three weeks later the patient was able to walk about outdoors, with two canes, and well able to bear weight on the leg. When he returned to Finland in October, six months after the operation, the fracture was consolidated and he walked well.

Case 7.—A 34-year-old soldier sustained a complicated femoral fracture through a shell injury in December, 1941. On admission to the Sahlgrenska Hospital, February, 1943, the wound was healed but the bone was not stable. Medullary-nailing was performed, March 27, 1943, after freshening of the bony ends and excision of all fibrous tissue. Firm fixation was obtained and full stability at the site of fracture. Two weeks later the patient was able to stand on his leg. After another two weeks he walked with

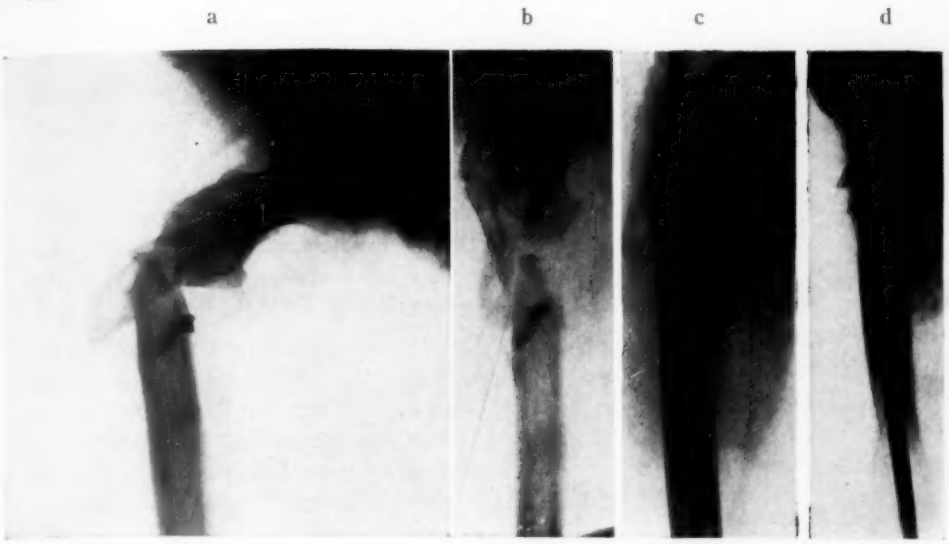


FIG. 6.—Case 7: (a) Pseudarthrosis femoris. (b) After reposition. (c) After marrow-nailing. (d) Three months later.

two canes. When he was discharged three months after operation, the bone was stable, he could walk well and there was increased callus formation. Report from Finland says that full consolidation has occurred (Fig. 6).

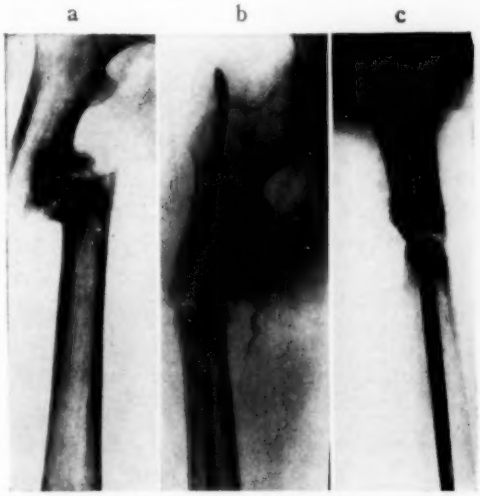


FIG. 7.—Case 8: (a) Pseudarthrosis of the femur. (b) After marrow-nailing. (c) Four months later.

Case 8.—In March, 1940, a 27-year-old soldier was hit by a shell splinter which caused a complicated femoral fracture. He was treated with extension and a plaster encasement. On admission to the Sahlgrenska Hospital in February, 1943, three years after the fracture, pseudarthrosis had developed. The leg was considerably shortened, the knee ankylotic, and there was paralysis of the peroneus muscle, with severe muscular atrophy. The patient was not able to stand on his leg at all. In February, osteosynthesis with bone transplantation was done. There being no signs of healing, the pseudarthrosis was revised again two months later, with excision of all the fibrous tissue and freshening of the bony ends, and medullary-nailing was done. Firm fixation and freedom from pain were obtained immediately, and the patient, who had lain in bed for nearly three years, was able to begin to bear weight on his leg after about a week. When he returned to Finland, three and one-half months after the operation he stood well on his leg, and walked with two canes. The bone was absolutely stable, but there was only a slight increase in the callus formation (Fig. 7).

In six cases of pseudarthrosis of the thigh bone which persisted from one and one-half to three years, and in spite of attempts with many different meth-

ods, it was not possible to attain osseous healing. In several of the cases all our old possibilities were exhausted and it is probable that the patients would have been left with the pseudarthrosis for the rest of their lives if we had not resorted to medullary-nailing. Also, it was a great mental relief to these invalids to be rid of pain and be able to get up soon after the nailing. Two of them had almost continuously been confined to bed for two or three years. In all cases the nail was inserted from the upper surface of the trochanter, and before insertion the wound was revised, with excision of fibrous tissue and freshening of the bony ends. As in these cases the fracture was exposed it was not difficult to get the fragments into such a position that the nail came into the marrow cavity of the distal fragment. In two of the cases, persisting fistulae, with a slight discharge, were present at the time of the operation. In one case the fistula healed soon after the operation, in the other there was still slight suppuration at the time the patient was discharged. In order to avoid the risk of reactivation of a latent infection sulfathiazole was in all cases introduced into the operation wound. The postoperative treatment was the simplest imaginable. After one or two weeks in bed the patient was allowed to get up. The prolonged stay in bed before the operation had produced more or less marked stiffness in the joints as well as muscular atrophy, necessitating intensive physical therapy.

Regarding the permanent results, in at least three cases out of six, consolidation had developed before the patient was discharged. The three others returned home so early that the final results could not be judged. There is much to indicate, however, that osseous healing will take place in these cases, as well, within reasonable time. Even if the results may not be 100 per cent perfect, experience, up to the present, indicates that the new method is vastly superior to the old ones for treatment of femoral pseudarthrosis.

Wherein *do the advantages of marrow-nailing lie*, and why is it that it has such a favorable effect in cases of pseudarthrosis? The most important condition for osseous healing is, of course, absolutely firm fixation of the fragments, and particularly firm fixation is just what is obtained with medullary-nailing, especially in cases of pseudarthrosis with sclerosed bone. Another important factor is early weight-bearing and after marrow-nailing the patient can begin weight-bearing on his leg almost immediately.

B. OTHER TYPES OF PSEUDARTHROSIS

Case 9.—A 25-year-old Finnish soldier was injured in the arm by a shell splinter in August, 1941. A defective humerus pseudarthrosis followed. After repeated revision, osteosynthesis was performed, September 7, 1942, but no consolidation resulted. Medullary-nailing was, therefore, done on June 11, 1943. The nail could not be placed centrally into the peripheral stump, and, consequently, the site of the fracture afterwards was not stable. Another nail was driven in on October 26, after extraction of the former one; nor was this nail inserted in the ideal position. To increase the stability a piece was taken from one of the tibiae and placed over the fracture. The wound healing was primary, and good stability resulted. Osseous healing has later occurred.

Case 10.—A 21-year-old Finnish soldier sustained a complicated fracture of the right upper and forearm in February, 1943. In both places pseudarthrosis developed. He arrived at the Sahlgrenska Hospital in June; and on June 22 medullary-nailing was performed to the humerus pseudarthrosis after excision of the fibrous tissue. The nail could not be placed in the desired central position, and full stability was not obtained. The arm was, therefore, placed in a plaster encasement. On November 12, a medullary-nail was inserted in the radius, as no stability had been obtained in spite of prolonged conservative treatment. This bone was fractured in two places and the nail was driven in from the distal end of the radius through the intermediate fragment, a long way into the proximal one. Excellent stability resulted (Fig. 8). However, slight suppuration occurred afterwards. When the patient returned to Finland in January, 1944, the fracture in the humerus felt consolidated, roentgenograms showed only moderate callus formation, and the suppuration in the forearm had almost entirely dried up.

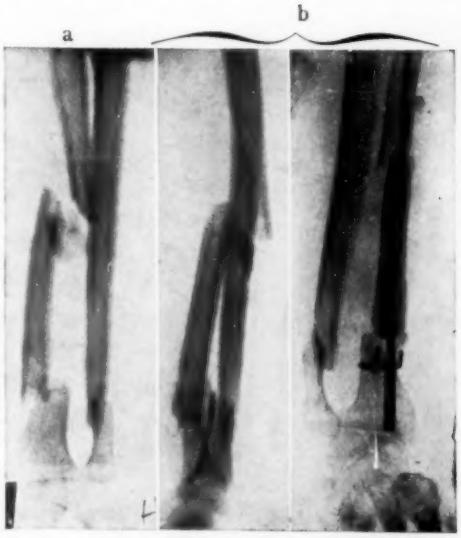


FIG. 8.—Case 10: (a) Pseudarthrosis of the radius in two places. (b) After nailing.

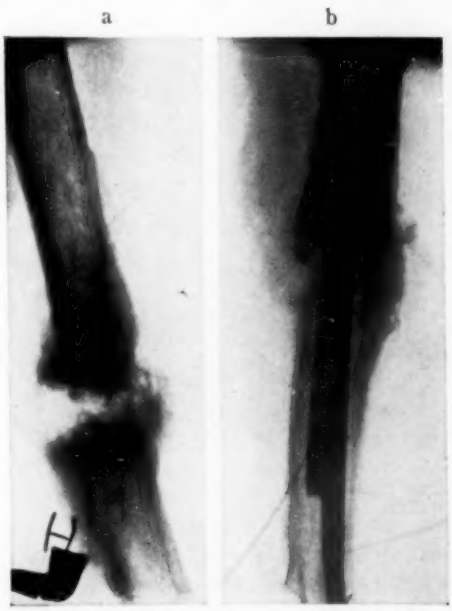


FIG. 9.—Case 11: (a) Three-year-old pseudarthrosis of the humerus. (b) Four months after marrow-nailing.

Case 11.—A woman, age 70, sustained a fracture of the right humeral diaphysis on October 17, 1941. Osteosynthesis with cerclage was performed. The threads cut in and typical pseudarthrosis developed. On July 27, 1942, the bony ends were revised and a large amount of bony shavings were transplanted, in accordance with the method of Levander. No healing resulted, and a new operation was performed on January 8, 1943, with transplantation of a bony plate from the tibia over the place of pseudarthrdosis. The fracture still did not heal. On May 15, two fairly narrow nails were inserted. Roentgenograms in October showed that the nails had broken in the middle of the pseudarthrosis. On October 22, the broken nails were extracted and two coarser ones were inserted after revision of the pseudarthrosis with removal of the fibrous connective tissue. Full stability was then obtained and, on February 1, 1944, roentgenograms showed that osseous healing had occurred three months after the last medullary-nailing. The nail was later removed (Fig. 9).

This case is of the greatest interest. The 70-year-old woman with a three-year-old pseudarthrosis of humerus shows osseous healing three months after marrow-nailing. Before we had tried all our old methods to obtain bone healing, such as osteosynthesis, bone transplantation, *etc.*, to no purpose.

Case 12.—A man, age 35, sustained a complicated fracture in his lower leg in February, 1942, resulting in prolonged suppuration and pseudarthrosis. In October, 1943, medullary-nailing was performed after chiseling off the end of the fibula, excision of the

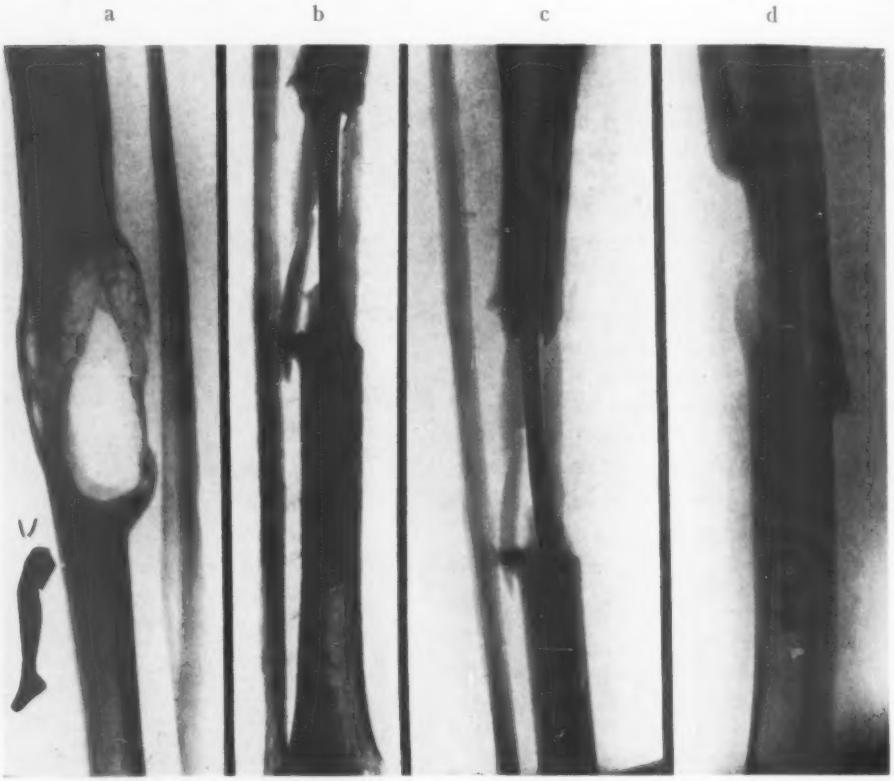


FIG. 10.—Case 13: (a) Cystic giant cell sarcoma of the tibia. (b) After resection, bone transplantation and marrow-nailing. (c) Four months after operation. (d) Twenty months after operation.

fibrous tissue in the pseudarthrosis and chiseling-off of the tibial ends. Plaster was applied. The wound healing was primary, and good stability was obtained. Six weeks later the leg could be weight-bearing. Full consolidation after four months.

It is more difficult, in cases of pseudarthrosis, to do the nailing, than in cases of recent fractures. The sclerosis in the end of the bone offers powerful resistance and may even make it impossible to insert the nail. Thus, in one case, not reported herein, it was not possible to drive the nail through the strongly sclerotic bone (tibia), and medullary-nailing could not be carried out. The inserted nail sat so firmly fixed in the bone that it could not be drawn out, and a piece of it had to be left there.

C. BONE PLASTIC

In the cases where there is a question of shortening or lengthening a leg, or to cover a deficiency of a bone, marrow-nailing is very useful, especially as the nail gives stability to bone and transplantation. The following cases illustrate this. The cases speak for themselves, and further comments are unnecessary.

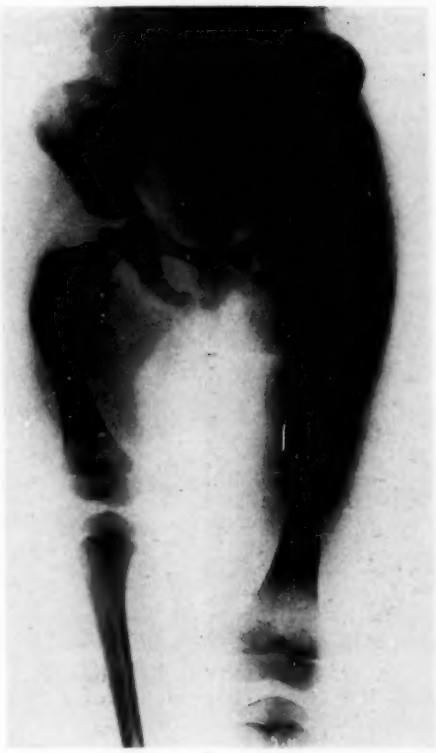


FIG. 11.—Case 14: Showing the type of congenital deformity.

Case 13.—Man, age 27, has been suffering from pains in left lower leg. Through misplacing his step, November 27, 1943, a severe pain in the leg developed. Roentgenograms showed fracture through a cystic tumor in the middle of the lower leg. Test extirpation from the cystic tumor showed osteitis fibrosa, with giant cell sarcoma, April 1, 1944. Resection of about 10 cm. of the tibia. Fixation of the bone ends by means of a large-sized medullary-nail. Intraplantation of two 9–10 cm. bone pieces taken from the right tibia. Plaster encasement.

January 25, 1946: The patient had not been examined since October, 1944, due to illness of nerves. Examination shows that both legs are equally long. No instability at the site of operation. Normal movability in knee and foot joint. Good weight-bearing capacity. Roentgenograms show good healing of the bone transplantation, in spite of the fact that the nail had broken. A small, narrow crack in the bone transplantation. Injury from tramcar accident on January 14, 1946 (Fig. 10).

Case 14.—Female, age 12. The right leg, as a result of a congenital disturbance in the growth, is 20 cm. shorter than the left one. The difference seems to be constant. On August 23, 1945, a 4-cm.-long piece was sawn out from the upper part of the left femur. Fixation of the fragment through a marrow-nailing. The piece of bone which had been taken from the left femur, intended to be used for lengthening of the right leg, was now left to remain between the bone and the muscles fixed to the periosteum (Fig. 11).

We dared not, at this same time, make intraplantation on the right side, fearing that this would be too much of a strain on the patient; and the double nailing would also have increased the risks for fat embolism. On October 3, a new operation with intraplantation of the piece from the left femur into the upper part of the right femur was undertaken (Fig. 12). As it was not possible to pull the bone pieces sufficiently apart to permit the insertion of the entire 4-cm.-long bone piece, 1 cm. had to be sawn out. The lengthening was, therefore, only 3 cm. Through these operations the difference between the legs was reduced from 18 to 11 cm. The healing progressed satisfactorily, and, already, four weeks after the last operation, the patient was allowed to rest on her left leg, and after four more weeks she was able to bear weight on her legs. We are thinking of making a similar operation upon the lower part of her legs after six months or a

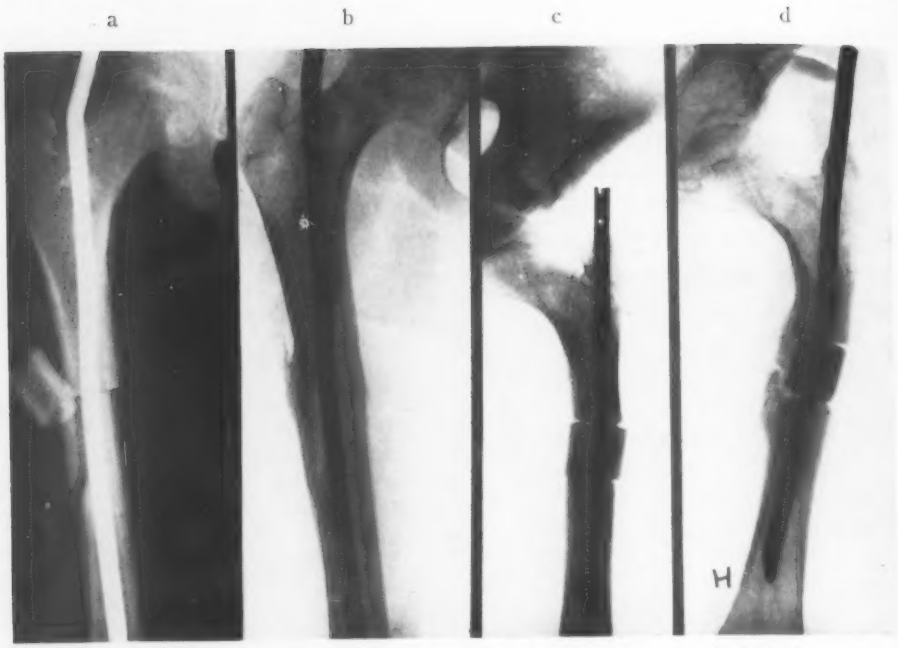


FIG. 12.—Case 14: (a) Resection of 4 cm. of the left femur. (b) Left femur six months later. (c) Intraplantation of a 3-cm.-long piece from the left femur. (d) Four months later.

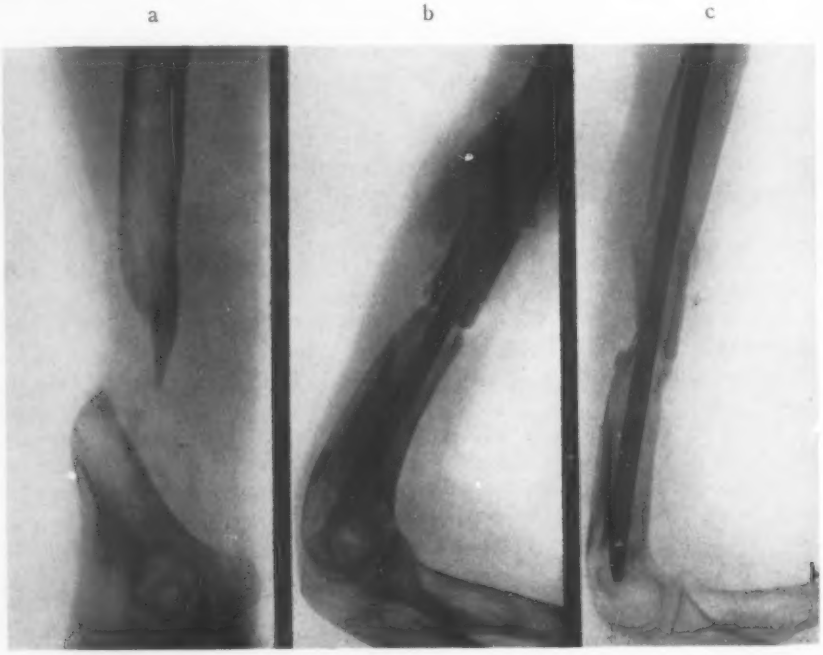


FIG. 13.—Case 15: (a) Defect pseudarthrosis of the humerus. (b) After bone transplantation and marrow-nailing. (c) Five months later.

year. Then the difference in the length of her legs would only be 5–6 cm. After a possible third operation her legs might become of equal length.*

Case 15.—Finnish soldier, age 29. After shell injury, August 5, 1944, prolonged suppuration and loosening of bone splints. When he entered the Sahlgrenska Hospital in August, 1945, the wound had been healed for two months. Roentgenograms showed a defect pseudarthrosis, with more than 5 cm. distance between the bone ends.

Operation.—August 30, 1945: After excision of all fibrous tissue and freshening of the bone ends, a 25-cm.-long marrow-nail was driven into the bone marrow. The bone defect was filled out by means of a 6-cm.-long piece of one of the fibulae. Very firm fixation resulted. A mild suppuration arose which soon ceased after penicillin treatment. Roentgenogram, in January, 1946, shows that the bone piece is healing in (Fig. 13).

CONCLUSIONS

In conclusion, I should like to express my opinion that *medullary-nailing constitutes a very great advance for recent fractures, as well as for pseudarthrosis and bone plastic.* In the treatment of fractures it can already, now, be said that this so-called *"stable osteosynthesis" is in many ways superior to the old methods of treatment,* particularly, the old forms of osteosynthesis. In my opinion, it will involve an equally great revolution in the treatment of fractures in the femoral diaphysis, as the method of nailing in the femoral neck did to the treatment of these fractures. In cases of pseudarthrosis and bone plastics marrow-nailings give such a firm fixation of the bone pieces that their healing in is made considerably easier. As plaster need not often be applied, stiff joints are avoided in several cases.

* In 1946 we did a new similar transplantation and the difference in the length of her legs is now 8 cm.

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CARDIAC RESUSCITATION

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SUDDEN CESSATION of cardiac activity is an emergency, the recognition and treatment of which are poorly understood by many surgeons. Inadequacy of management of this catastrophe is common in the practice of general surgery, but even more so in the surgical specialties in which resuscitation is rarely achieved. Prompt diagnosis, courageous decision and intervention based on a rehearsed plan of action can prevent fatalities which at the moment seem inevitable. Experience with four patients in whom cardiac arrest occurred, and in whom restoration of the heart beat was accomplished leads us to re-emphasize the teachings of such pioneers as Bailey[1, 2] Beck,[3] Wiggers[4] and others who have stressed the basic principles on which treatment is founded.

Two problems demand attention. The heart beat must be restarted, and the central nervous system must not be deprived of oxygenated blood for more than three or four minutes. Cerebral anemia of greater duration is followed by widespread cerebral cortical destruction and death. This sequence of events occurred in two of our patients. The other two patients were returned to normal health.

DIAGNOSIS

The primary cause of failure in cardiac resuscitation is delay in diagnosis. During intrathoracic operations the heart can be directly visualized and recognition of cardiac inactivity is easy. If a major artery such as the aorta, the carotid, brachial or femoral can be inspected or palpated through the operative wound the status of the heart beat can also be readily determined. Under other circumstances, however, when no peripheral pulse is palpable and blood pressure cannot be obtained by auscultation, three possibilities must be considered: (1) the heart has stopped beating entirely, (2) the ventricles are fibrillating or (3) cardiac contractions are so feeble that insufficient blood is ejected to raise arterial pressure to the level at which a peripheral pulse can be felt. The majority of patients are doomed because minutes which cannot be spared are wasted in searching for a stethoscope, in frantic palpation of a succession of arteries, in hasty consultation with colleagues summoned to judge a situation with which they are unfamiliar.

The only certain method of ascertaining whether the heart is still beating is to palpate or visualize the heart or large arteries directly. A second, less

dependable procedure, is to visualize the retinal vessels through an ophthalmoscope (in the absence of cardiac contractions the retinal arteries will not be visible (5) and the column of blood in the veins will be broken up into short segments.) Theelectrocardiographic tracing unfortunately cannot be relied upon. The work of Negovski indicates that at the time of cardiac arrest the heart current can still (6) produce for some time an electrocardiogram with deviations from the normal. The absence of capillary refill is strong evidence that the circulation has stopped but may be difficult to evaluate particularly in dark skinned individuals.

The implications of the above are clear. Opening the thorax is the only conclusive *diagnostic* procedure. If the heart has stopped or the ventricles are fibrillating minutes can be saved by adopting the attitude of rapidly opening the chest for diagnosis, whereas if the heart is beating feebly little harm may have been done.

TREATMENT

Artificial ventilation of the lungs with 100 per cent oxygen and cardiac massage are the essentials of therapy.

The method of artificial respiration depends upon the circumstances. If cardiac arrest has occurred in the operating room, an anesthesia machine is satisfactory. Manual compression of the breathing bag will inflate the lungs and the elastic recoil of the respiratory organs completes the cycle. If resuscitation is attempted elsewhere the bag and mask technic[7] or the Kreiselman "bellows" resuscitator[8] are simple and effective. One should guard against excessive intrapulmonary pressures since such pressures will reduce venous return to the heart by blocking blood flow through the lungs.[9, 10]

Although respiration (normal or artificial) can cause some blood to circulate, the most effective mechanism for movement of blood is the pumping action of a contracting heart. In the absence of spontaneous cardiac activity manual compression of the ventricles will provide sufficient circulation to maintain the integrity of the central nervous system until rhythmic cardiac contractions are re-established. Adequate exposure of the heart is therefore essential and must be obtained at the earliest possible moment.

A transverse incision in the fourth left intercostal space is best. The fourth and fifth ribs can readily be spread apart and the heart grasped by the operator. Exposure may be increased by dividing the fourth and fifth costal cartilages. A direct approach through the chest wall is superior to an abdominal incision with an attempt to reach the heart through either an intact or an incised diaphragm. Opening of the pericardium is not required.

The heart is compressed firmly at the rate of 20–40 times per minute, depending upon the adequacy with which the ventricles fill between compressions. Each compression raises arterial pressure 60–70 mm Hg. and a pulse can often be felt in a peripheral vessel. To increase blood flow through the coronary arteries the aorta may be occasionally compressed just above these vessels.[4]

The role of epinephrine in cardiac resuscitation is uncertain. In the experimental laboratory when cardiac action has ceased it is common practice to inject this potent cardiac stimulant into the heart chamber, massage the heart and achieve a successful result. Theoretical objections to the drug exist, however, since increased myocardial irritability and the development of ventricular fibrillation are known to follow its administration. If epinephrine is to be used, it should be placed into the auricle rather than the ventricle. This confines any abnormal stimulus (chemical or mechanical) to a chamber in which fibrillation is of little significance.

Ventricular fibrillation. If circulatory collapse is due to fibrillation of the ventricles rather than to complete cessation of cardiac activity, a different type of therapy must be employed.

The most effective method of treatment is the technic of serial defibrillation developed by Wiggers.[4] An electric current of 1.0 ampere (60 cycle) is passed through two brass discs 2–3 inches in diameter which are applied to the sides of the heart. Each shock lasts less than one second; 1–2 seconds elapse between shocks; 3–7 shocks as a rule suffice. Wiggers' plan is to merge small fibrillating areas into larger ones, and then stop the entire fibrillation process with one final electric shock. Electrodes can be kept sterile in operating rooms, and application is simple once the heart is exposed. Before counter shocks are thrown into the heart, it should be manually compressed for 30-60 seconds. This provides blood for the myocardium and may improve the chance of recovery. Countershock therapy is successful in a high percentage of instances. Once fibrillation has ceased, of course, one is again faced with the problem of re-starting rhythmic cardiac activity.

The use of procaine to prevent fibrillation and to treat it once it has developed has been suggested. There is no doubt that procaine raises the threshold of the heart to fibrillation produced by epinephrine.[11, 12, 13] It has also been shown that the intravenous or intracardiac injection of procaine into anesthetized dogs which have developed cardiac arrhythmias with a shift of the pacemaker has caused a return of the pacemaker to the sinus node.[14] Finally, during cardiac surgery in World War II experience was gained which suggested that results on animals might be applicable to man. Burstein[15] injected single doses of 30–70 mg. procaine intravenously into 14 anesthetized patients with acute arrhythmias occurring during intrathoracic surgery. No untoward effects on the nervous system, respiration or circulation were observed. The dysrhythmias always improved, often dramatically. Wiggers[13] on the other hand, although admitting that procaine raises the fibrillation threshold of the ventricle, states that the drug is not a preventive and believes that it is unwise to use depressants such as local anesthetic agents, or to attempt to modify conduction. Stutzman *et al.*[16] also were unable to reverse ventricular fibrillation with intravenous or intracardiac injections of procaine made within 30 seconds of the onset of the fibrillation. The question is therefore not settled and further work must be done to evaluate the myocardial

depressant action of this local anesthetic. It must be realized that the intravenous injection of procaine without cardiac massage can accomplish nothing if the ventricles are fibrillating, since the drug cannot reach the heart.

Adjunctive therapy. Successful cardiac resuscitation usually leaves one with a patient who is unconscious for a certain period of time. Such a patient must receive constant care if secondary complications are to be avoided. Secretions which accumulate in the pharynx and tracheobronchial tree must be aspirated. Prophylactic injections of penicillin are often advisable to minimize pulmonary infection. The patient must be turned frequently from side to side to avoid hypostatic congestion in the dependent portions of the lungs. Urinary output must be maintained by an adequate fluid intake, and constant attention must be paid to the bladder. Inhalation of oxygen is employed, although it is uncertain whether this is helpful in the presence of a normal arterial oxygen saturation. Blood volume and hematocrit determinations are of value in deciding whether blood or plasma transfusions should be used. All of these measures were used in the management of the patients reported in this paper.

PROGNOSIS

1. What is the likelihood that the heart can be started again?

The heart is an amazingly resistant organ, possessing rhythmicity and contractility which are difficult to destroy. Kountz[17] studied the hearts of individuals who had died five minutes to six hours prior to his examination. By perfusing the coronary arteries he was able to revive 58 of 127 hearts. Forty-eight of these beat rhythmically for at least two hours. Of 34 hearts which he observed within 30 minutes after death, 24 were revived. Of 29 others studied within 30–60 minutes, 17 resumed regular contractions. He was able to restore only four of the 20 hearts perfused five to six hours postmortem.

Whether the heart can be made to beat again regularly, and whether this activity can be maintained depends upon the age of the patient, the cardiac status prior to the catastrophe and the cause of the cardiac arrest. Young, healthy hearts can be restored to normalcy with relative ease, but a myocardium damaged by toxemia, arteriosclerosis, hypertension, coronary insufficiency, or narcosis is much less likely to recover.

In general, if prompt diagnosis is followed by prompt therapy, the heart can be started again. If, on the other hand, there is hesitancy rather than boldness, or if intravenous therapy is attempted before attention is directed towards the heart, the myocardium may have been sufficiently damaged by anoxia to resist all efforts at resuscitation.

2. If the circulation is restored, what will be the status of the patient's central nervous system?

When the heart returns to a regular rhythm, and blood pressure is maintained, spontaneous respiratory activity can be expected to reappear within 5 to 30 minutes. This indicates that medullary cells are the most resistant

in the central nervous system, a fact clearly established by many investigators. This also illustrates the fact that in the medulla itself the vasomotor center is more resistant than the respiratory center.

Once vital functions have been restored, two possibilities exist. (1) The patient may steadily improve until consciousness is regained and a partial or complete recovery is made. Unless the period of circulatory cessation has not exceeded two to three minutes the postoperative course is stormy. Or (2) consciousness is never regained and death occurs. The heart may again cease to beat within a few minutes or hours, or the fatality may be delayed for weeks (26 days in one case reported in England[18]).

The duration of cerebral anoxia determines the prognosis. Quantitative data are not available for man, but the work of Weinberger, Gibbon & Gibbon[5] in cats, gives an index of the ability of the mammalian central nervous system to withstand complete deprivation of blood supply. These investigators clamped off the pulmonary artery suddenly stopping blood flow throughout the body. The clamp was released at various times and the degree of recovery was correlated as follows:

Duration of Circulatory Arrest	*Recovery*
1. 2 min. to 3 min. 10 sec.	Complete within 24 hours.
2. 3½ min. to 5¾ min.	Wildly excitable for 24 hours; gradual improvement until at end of one week there remained only alterations in behavior and loss of normal intelligence.
3. 6 min. to 7¾ min.	Only three of nine animals survived. The typical picture was that of hyperactivity and wild purposeless behavior, followed by stupor or indifference to stimuli. The cortex suffered irreparable damage. There was blindness, spasticity and dementia. The animals who survived were little more than reflex mechanisms.
4. 8¾ min. to 10¼ min.	All seven animals in this group died within 75 minutes to 48 hours of the circulatory arrest. Coma, alternating with convulsions, was characteristic of the brief survival period.

Certain factors may increase the resistance to anoxia. For example, anesthesia may protect by depressing cellular activity, and reducing the demand for oxygen per unit of time. This has been demonstrated by Rosenthal, Shenkin and Drabkin[19] in carbon monoxide poisoning in which 75 per cent carbonyl hemoglobin was found to be the critical level for *unanesthetized* dogs. Lesions of the brain were always seen in dogs kept for brief periods under these conditions. Dogs under light surgical anesthesia with nembutal, however, survived, without ill effects, acute carbon monoxide hypoxia at levels as high as 85 per cent (and in one instance 93 per cent) of HbCO in the blood. Furthermore, if cardiac arrest occurs during the course of closed system anesthesia and high oxygen concentrations have been used, the extra oxygen dissolved in the plasma may prolong survival of the central nervous system.

CASE REPORTS

Case 1.—A. S., a 51-year-old Negro male, had a toxic nodular goiter for at least five years prior to admission. For three years he had had auricular fibrillation and, because of thyrocardiac symptoms, was a virtual invalid. Repeated courses of iodine and thiouracil resulted in incomplete remissions but he refused operation until the present admission. During the period of preoperative preparation he was digitalized, and gained weight satisfactorily. The risk of operation was not considered unusually great.

On 3/3/47, the morning of operation, he was given morphine 10 mg., scopolamine hydrobromide 0.3 mg., and atropine sulphate 0.4 mg. at 7:30 A.M. At 7:50 A.M., 10 cc. of a 5 per cent solution of pentothal were administered rapidly and an endotracheal tube was inserted. Anesthesia was maintained with a slow drip of 1 per cent pentothal sodium and inhalation of a 70 per cent nitrous oxide, 30 per cent oxygen mixture through a partially closed system. During the first 25 minutes of the operation the surgeons thought the blood was abnormally dark. The anesthetist did not believe it was darker than normal venous blood and considered that there was no evidence in the pulse rate or blood pressure of oxygen deficit. Substitution of cyclopropane for nitrous oxide resulted in a much lighter color of the blood. The blood pressure promptly rose from 140/80, which it had been since the beginning of the operation, to 210/90, then to 230/90, and the pulse became more irregular. With resumption of the nitrous oxide-oxygen mixture the blood again became dark and the blood pressure gradually fell, during the next 15 minutes, to 110/80, with a pulse rate of 78.

Fifty-five minutes after the operation was begun the temporal pulse, which was being constantly palpated, suddenly disappeared. This occurred between 8:59 and 9:00 A.M. Approximately 4 minutes later the chest was opened and the diagnosis of cardiac arrest was confirmed. After thirty seconds of cardiac massage the heart began to beat spontaneously and the blood pressure was 100/70. One minute later the ventricles began to fibrillate and the heart was again massaged. Two cc. of 1 per cent procaine were injected into the pericardium and 3 cc. into the right ventricle. Bronze disc electrodes were applied to the heart and after the third electrical shock (2 amp) which caused a violent spasm of the entire body, ventricular fibrillation stopped and normal spontaneous ventricular rhythm resumed (9:15 A.M.) although the auricles continued to fibrillate. Less than one minute later spontaneous respirations (24–30 per minute) began and the blood pressure was 140/80. Resection of the right lobe had just been completed when cardiac arrest occurred. The wound was quickly closed. The total dosage of 1 per cent pentothal sodium was 65 cc.

At 9:37 A.M. a swallowing reflex was noted and at 10:30 A.M., during tracheobronchial toilet and removal of the endotracheal tube, the cough reflex appeared quite active. The pupils were quite small and reacted sluggishly to light.

After resumption of spontaneous cardiac activity the pulse rate and blood pressure remained remarkably constant, the pulse ranging from 80 to 110, and the blood pressure from 120/80 to 110/95. At 11 A.M. the rectal temperature was 102° F. Fearing hyperthermia, an ice pack ws applied from toes to chest. This soon caused violent shivering and peripheral cyanosis, but reduced the temperature to 99° F. Whenever the rectal temperature rose to 103° F. ice was reapplied and this was always accompanied by shivering and cyanosis. The repeated use of ice packs was effective in keeping the rectal temperature below 104.5° F.

An electrocardiogram at 11 A.M. showed a right bundle branch block, with inverted P waves in leads 2 and 3, and in all chest leads. At 12:45 A.M. another electrocardiogram showed frequent auricular extra systoles, and almost complete disappearance of the bundle branch block. The T waves were upright in the limb and chest leads.

On neurologic examination at 12:30 P.M. there was no spasticity but all deep reflexes were hyperactive, and there were positive Hoffman and Troemmer reflexes. Plantar responses were neutral, abdominal reflexes were absent, and there was no clonus.

At 7:00 P.M. brief clonic convulsions involving both lower extremities began and an hour later there were violent, generalized clonic convulsions. These continued to occur at frequent intervals, ranging from a few seconds to several minutes in duration. The cerebral spinal fluid pressure was normal and the fluid was clear. About midnight Cheyne-Stokes respirations were noted and thereafter recurred at frequent intervals.

During the next four days the picture remained essentially the same, *i.e.*, a comatose patient, with intermittent generalized clonic convulsions and Cheyne-Stokes respirations. The pupils reacted sluggishly to light. At times a corneal reflex was noted and on a few occasions pressure on the supraorbital nerves resulted in slight movement of the right arm. Accumulation of tracheobronchial secretions required more and more frequent aspirations. One millions units of penicillin daily were given prophylactically. Fluids, electrolytes, and vitamins were given parenterally and the urinary output was more than 1500 cc. daily. On the night of the fourth postoperative day he was obviously moribund and death occurred at 10:30 A.M. on 3/8/47.

Case 2.—B. A., a 21-year-old female with gangrenous appendicitis. An appendectomy was begun at 1:30 A.M., 4/4/46 under procaine-pontocaine spinal anesthesia. This patient weighed 190 lbs. and was only 5′1″ tall. Her sensory level of anesthesia rose rapidly to the sixth cervical dermatome. This was accompanied by inability to talk, respiratory depression and finally acute circulatory failure, with no obtainable blood pressure. The full significance of the situation was not appreciated by the anesthetist for a few minutes at least, so that it is difficult to determine the exact duration of cardiac arrest with certainty. This is estimated at six to eight minutes at a minimum. Therapy consisted of an intracardiac injection of desoxyephedrine, and artificial ventilation with 100 per cent oxygen. Cardiac massage through the diaphragm was then instituted and continued for eight minutes before spontaneous cardiac contractions began. With each cardiac compression an impulse could be palpated in the radial artery. With the return of rhythmic action the blood pressure was 102/80, pulse rate 120. Spontaneous respiration was noted in 30 minutes. Soon after breathing was re-established twitching of the arms and legs commenced. The operation was completed at 3 A.M. Blood pressure at the end of the operation was 80/50, pulse 140.

Four hours postoperatively rectal temperature had risen to 108° F. and the twitchings described above had progressed to severe convulsive movements of the entire body. Ice water sponges and ice water enemata together with sodium luminal 60 mg. intramuscularly were administered and an electric fan was set up to blow over the patient's body. The twitchings were reduced in frequency and intensity and the temperature fell to 102° F. At this point the patient responded to painful stimuli by moaning and by moving her extremities.

At the sixth postoperative hour the blood pressure was 70/50, temperature 104° F. and the patient was given 150 cc. of 50 per cent glucose by vein. This was administered in an attempt to decrease the cerebral edema thought to be present following severe anoxia whether by coincidence or on a cause-effect relationship. The response to this therapy was dramatic. The patient became much quieter, the temperature fell to 99° F., the pulse rate fell from 120 to 80 per minute and the blood pressure rose to 114/58. The general appearance was more that of sleep. At the eighth postoperative hour projectile vomiting began and the patient moaned and moved about in bed. Neurologic examination at this time revealed a positive Babinski reflex on the right side, frequent contractions of the extremities, eye balls rolled up, pupils reacting to light. Twelve hours postoperatively it appeared as though the patient might recover. She seemed only semistuporous and responded to her name. Considerable difficulty was had in maintaining an adequate blood pressure and for the greater part of this first postoperative day her blood pressure ranged between 70 and 90 mm Hg. systolic. The usual anti-shock measures were employed. Estimates of plasma volume at this time indicated a volume of

3200 cc. which was about 500 cc. below her supposed normal. An hematocrit of 33 per cent was obtained.

The next problem which presented itself was that of mucus in the tracheobronchial tree. Frequent aspiration was necessary and finally an endotracheal tube was inserted and considerable amounts of blood-stained mucus were aspirated frequently. The patient's color remained good, vital signs were adequate and because of her apparent response to relatively slight stimuli it was felt that she would survive. A neurologic examination made 12 hours postoperatively (Dr. A. M. Ornsteen) was reported as follows: "Sensorium clouded, mildly reactive to pain stimulus, blinking active with eyes open, probably not conscious of light stimuli. Pupils 3 Mm. react well and equally. Fundi normal. Trismus; neck free; general flexor spasm of distal portion of limbs. Spinal reflexes of defense marked. Fragmentary tonic neck reflex, as head moved to right the right arm came up, no pathologic reflexes. The present picture is that of mesencephalic edema, secondary stasis and perivascular diapedesis."

During the next 24 hours the patient became less reactive. She responded less to stimuli. At the same time it was evident that despite frequent tracheal aspirations there were many coarse rales which remained in the larger bronchi. She was bronchoscoped and thoroughly aspirated. In spite of an adequate fluid intake urine volume was small.

Cyanosis and labored respiration were first noted 48 hours postoperatively. This was associated with a sudden decrease in blood pressure and with spasticity and convulsive seizures. The rectal temperature, which had been slowly rising, reached 106° F. It became obvious that serious cerebral damage had occurred and that if the patient survived she would be decerebrate. She was in deep coma. During the remainder of her course she had recurring bouts of cyanosis, labored respiration and hypotension. She died of pulmonary edema 64 hours postoperatively. Autopsy revealed pulmonary edema as the only gross finding.

Case 3.—M. L., age 20, with mediastinal tumor. An exploratory thoracotomy was planned, and anesthesia with cyclopropane-oxygen was begun at 12:50 P.M., 6/1/45. Because of a gross overdose of the anesthetic agent, or as the result of an inhibitory reflex secondary to endotracheal intubation,[22] heart action ceased at 1:01 P.M. One cc. of 1:1000 epinephrine was injected into the right ventricle and 100 per cent oxygen was administered through the endotracheal tube. Since epinephrine caused no response, the patient was moved into the operating room, the thorax was opened under aseptic technic and cardiac massage of a motionless, flabby heart was begun at 1:06 P.M. Feeble contractions were noted after the third compression. Once started this cardiac activity increased steadily in vigor until full rhythmic beats were evident. Blood pressure rose to 140/100. Spontaneous respiration was noted 25 minutes after the heart started to beat again.

At 2:15 P.M. limb reflexes were hyperactive but no abnormal reflexes were elicited. The patient moaned occasionally and moved her extremities about aimlessly. At 3:45 P.M. twitching of the limbs was first noted. These spasms, brief at first, progressed in severity and duration until at 4:15 P.M. there were rapidly recurring tonic bouts of rigidity involving the entire body. These episodes which lasted 60 to 90 seconds could be initiated by even the slightest stimulus. By 5:30 P.M. the rigidity was less intense and spasms occurred less frequently. At 7:00 P.M. some of the patient's movements appeared purposeful. She was no longer comatose, but was in a state of semi-coma.

By 9:00 P.M. the spasms of rigidity had almost ceased. In their place there occurred violent muscular activity in response to such stimuli as extension of the arm. Restlessness was marked and restraints were necessary. At 12:00 midnight the patient looked and acted like a wounded animal, screaming aloud, incoherently, and thrashing around in bed. Blood pressure was 120/96, pulse 108, respiratory rate 24, rectal temperature 101° F. Bouts of extreme activity alternated with periods of deep sleep. After each of

these rest periods she seemed on the verge of talking and could squeeze one's hand in response to a command. By 4:00 A.M. she was less restless and responded less violently to stimuli. Her fingers were held as though in tetany, or parkinsonism. Her arms were folded across her chest. At 8:00 A.M. she opened her eyes and at 9:00 A.M. spoke her name distinctly. She continued to sleep for the majority of the time.

At 2:00 P.M., 6/2/45, a neurologic examination (Dr. A. M. Ornsteen) was reported as follows: "The patient is somnolent. When aroused she co-operates well; attention is only fair, apperception poor. She localizes physical distress well. Left arm less used in gestures. Neither arm or either leg can be held up well. The upper extremities drift down, left more than right. Lower extremities not held up at all. Extraocular movements normal, no nystagmus, pupils normal, react to light, fundi negative. Hand grips are good considering the circumstances. Left ankle jerk is weak, right ankle jerk absent. Neither knee jerk obtained. Arm jerks present, right greater than left; normal plantar reflexes. No abdominal reflexes. Dysarthric but not asphasic." Mental confusion persisted. There were periods of complete irrationality and marked restlessness succeeded by profound exhaustion and deep sleep. When awake she spoke distinctly but with hesitation. She recognized her family and called them by their proper names.

June 3, 1945. The patient carried on an intelligent conversation but had retrograde amnesia and appeared euphoric. Twelve hours later this mental attitude was replaced by one of antagonism.

June 4, 1945. She was oriented for the first time, knew where she was but had no idea of recent past events.

June 5, 1945. Completely rational during the day, but at night she was seized with vivid hallucinations and episodes of disorientation. She was up in a wheel chair and although weak, she exhibited good motor control.

June 6, 1945. Began to recall events before her operation, knew for the first time where she was and why she was there.

June 8, 1945. Much clearer mentally, but very tired and irritable.

June 11, 1945. Neurologic examination was entirely negative. "No cerebellar or dysarthric signs. No subjective complaints. Extraocular movements normal, no nystagmus. No cranial nerve changes. Plantar reflexes normal. Mental status now normal. Consider her fully received." The patient was discharged on June 22, 1945. On September 19, 1945, a right pneumonectomy was performed by one of us (W. H. E.) and a malignant tumor of the right upper lobe was removed. Anesthesia and surgery progressed uneventfully on this occasion. A follow-up report on June 25, 1946, indicated that metastases had occurred and the patient was losing weight steadily. Mentally she continued to be normal.

Case 4.—Chao Da Thea, a 22-year-old Chinese soldier, was given a right brachial plexus block at 8:10 A.M., February 9, 1944. A supraclavicular approach was used. Aspiration before injection of the local anesthesia produced no blood. During a four-minute period, 16 cc. of 2 per cent metycaine in water with epinephrine were deposited over the trunks of the brachial plexus. At 8:14 A.M. the injection was stopped because the patient began to retch. Cyanosis of the lips was noted. Blood pressure was 120/60, pulse rate 108. One hundred per cent oxygen was administered by a B. L. B. face mask.

At 8:20 A.M. the pulse was still palpable. Two cc. of a 5 per cent solution of pentothal were injected intravenously. At 8:21 A.M. respiration ceased, nor could pulse or blood pressure be obtained. The lungs were artificially inflated with oxygen from an anesthesia machine. At 8:22 A.M. the heart was first examined with a stethoscope and no sounds were audible.

The chest was hastily prepared for an incision in the 5th left interspace. The heart was exposed at 8:25 A.M. It was in standstill. Artificial systole was begun manually. Rhythmic contractions appeared after 60 seconds of this therapy. At 8:40 A.M. spon-

taneous respiratory activity was noted, and 5 minutes later the patient began to move about on the operating table. The thoracotomy incision was closed at 8:55 A.M., at which time the patient was thrashing around wildly and groaning aloud.

For the next hour and one half, the patient exhibited the clinical picture of cerebral irritability resulting from acute hypoxia. He was manic and showed bizarre neurologic phenomena—atypical opisthotonos and spasticity of the flexor muscle groups. There was a rhythmic pattern to the convulsive activity, two or three minutes of wild excitement and generalized muscular rigidity being followed by moments of quiet and relaxation. Oxygen was administered intermittently throughout this period.

Ten cc. of a 5 per cent pentothal solution were administered intravenously from 10:20 to 10:30 A.M. in an attempt to control the hyperkinetic activity with light narcosis. At 11:00 A.M. the effects of this had largely disappeared. During the next four hours, the patient's general condition remained the same. There were alternating periods of motor restlessness and spasticity and quiet. He began to respond to external stimuli by opening his eyes. No hyperthermia developed. By 8:00 P.M. he had recovered consciousness and answered questions. He was still obviously confused. Rectal temperature was 100.3° F. The circulation was normal.

By the following morning, he had made a complete recovery, and there were no sequelae of any kind. Recovery was uneventful. The patient got out of bed on his fourth postoperative day. He was seen again six months later and was found to be completely normal.

COMMENT

The causes of cardiac arrest in the four patients discussed above were varied. In Case 1 many factors require consideration. Cardiac damage was already present preoperatively. Digitalis had been administered. This drug *per se* is believed to increase myocardial irritability. Anoxia was evident during part of the surgical procedure. The sudden rise in blood pressure ten minutes before the catastrophe may have placed a great load on the heart. Finally, the possibility of vagal reflexes arising as the result of manipulation around the thyroid gland should be mentioned. In all probability the combination of increased work, decreased oxygen supply, and increased irritability proved too much for a thyrotoxic heart.

In Case 2, a so-called total spinal anesthesia was unfortunately achieved. Paralysis of the muscles of respiration occurred together with a sharp reduction in blood pressure. Decreased coronary blood flow, and a diminished venous return of such magnitude that the heart muscle had little against which to contract would seem sufficient explanation for cardiac failure under these circumstances. In Case 3 an overdose of the anesthetic agent, or cardioinhibitory reflexes secondary to endotracheal intubation were probably responsible for cardiac arrest in an otherwise normal, young girl. In Case 4 one can postulate idiosyncrasy to the local anesthetic agent, or, more likely, inadvertent intravenous injection or rapid absorption of the drug. The depressant action of even a small dose of pentothal under these circumstances may have been contributory.

Faced with a heart which was not beating, the anesthetists and surgeons in each instance reacted with what might appear to be reasonable promptness. Cardiac massage was instituted and restoration of the heart beat was accomplished. "Reasonable promptness," however, was inadequate, for only two

of the patients made complete recoveries. In Cases 1 and 2 the delay of five to eight minutes before the heart was manually compressed was followed by irreversible anoxic changes in the cerebral cortex. The margin between a successful outcome and a fatality is indeed slim as these four experiences indicate.

If cessation of cardiac activity has been brief, convalescence may be indistinguishable from that seen in the average surgical patient, as illustrated by case reports of Lium[20] and Adams and Hand.[21] If the duration of cerebral anoxia has been slightly more prolonged, convalescence may be stormy, but with indications of steady improvement as each hour passes (Cases 3 and 4). Cases 1 and 2 on the other hand, are examples of the tragic decline of an organism which has suffered irreversible changes.

Acute febrile responses (102°–108° F.), manifestations of cerebral cortical irritability (muscular rigidity, twitching, convulsions, hyperflexia) and varying degrees of coma are characteristic. The time of onset and the duration of these phenomena are of interest since analysis of such data may permit prediction of the outcome of a particular case. Our experiences indicate that recovery can be predicted on the basis of events occurring within the first 12 hours. Persistence of coma beyond this period is unfavorable. It is to be hoped that this type of patient will be more accurately observed in the future, particularly from the neurologic standpoint. Much information of value can be obtained from such studies.

Certain problems raised in this paper demand investigation. It is apparent that the brain is the most vulnerable of all body tissues as far as oxygen deficit is concerned. Is there any means of protecting this organ preoperatively? Can a reserve of glucose, enzymes, vitamins or other essential elements be built up in the same way as the liver is protected by preoperative therapy? If this is impossible, can the need of the brain for oxygen and nutritive material be safely reduced in some way during operation so that in an emergency the time of survival of the central nervous system may be prolonged? Finally, since time is so important, should attention be directed at once towards perfusing the brain via the carotid arterial system before any other therapeutic efforts are made? Of the measures commonly adopted during the management of cardiac arrest epinephrine and procaine require further critical study. Both have clinical and experimental evidence suggesting their utility, yet theoretical objections to each are strong. The status of intra-arterial[23, 24] and intracardiac[25] infusions must also be defined. By immediately raising pressure throughout the arterial tree the former has distinct advantages over the intravenous route for administration of fluid and by providing a volume of fluid against which the ventricles can contract the latter may be useful.

Above all, however, is the necessity for a greater awareness of the possibility of cardiac arrest on the part of anesthetists and surgeons. Resumption of normal cardiac activity must be the first consideration, when deterioration of a patient's condition becomes evident.

SUMMARY

Prompt diagnosis and prompt therapy are essential for cardiac resuscitation. Treatment must be boldly executed according to a carefully worked out plan, consisting primarily of artificial respiration with 100 per cent oxygen and cardiac massage. The success or failure of such a program depends entirely on the length of time that the brain is without blood supply. If restoration of the circulation occurs within three to five minutes complete recovery can be expected, particularly in young, previously healthy individuals. If much more time than this elapses before resuscitation, permanent cerebral damage will occur. The course of four patients in whom cardiac resuscitation was successful is described.

Acknowledgement is made to Philip M. Gleason, M.D., who assisted in the treatment of Case 4.

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METABOLIC STUDY OF BURN CASES

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THE WORK TO BE DESCRIBED, which was the subject of a preliminary communication,[1] had as its principal object a complete study of the nitrogen balance of burned patients, including the loss of protein in the exudation from the burned area. For a time, however, ward facilities were not reliable for accurate metabolic work, and attention was therefore directed to simple determination of the nitrogen in the urine, measurement of the creatinine and creatine excretion, plasma protein estimations, and so on. While some of that work has been useful, it is unfortunate that many of the early figures obtained had to be disregarded, as examination of the ward records showed that no reliance could be placed on the collections of urine being complete or on their having covered the stated periods of collection. As stated in the preliminary note, this trouble was largely due to shortage of trained staff; and it is part of the purpose of this report to draw attention to the absolute necessity for the collection of specimens to be under the supervision of a reliable, responsible person, if the laboratory work in such metabolic investigations is not to be vitiated.

In certain cases, although the urine specimens were clearly not accurate 24-hour collections, it is possible to give a figure for the average daily nitrogen excretion over a period, as little or none appeared to have been lost.

Since then we were fortunate in securing the services of Nurse L. Thrussell for a short time (by the kindness of Professor R. A. McCance) and, more recently, of Sister R. M. Selley: they have looked after the patients on whom nitrogen balances were carried out and have supervised the feeding and the collection of specimens in these cases.

The report is divided into the following sections:

1. Urinary nitrogen excretion.
2. Nitrogen balances.
3. Nitrogen in the exudate from the burned area.
4. Plasma proteins.
5. Creatinine and creatine excretion.
6. Proteinuria.
7. Chloride excretion and plasma chloride level.
8. Blood sugar levels.

It was hoped to do some work on the nitrogen partition of blood and plasma and urine, in view of the large undetermined nitrogen values reported by some of the American workers;[2, 3] but unfortunately pressure of work prevented this.

The results of liver function tests in many of these cases are to be published by Mr. Garfield Thomas.

METHODS

Urine was collected in bottles with chloroform, or a 5 per cent solution of thymol in chloroform, as preservative.

Total nitrogen in urine was determined by the Kjeldahl method. Checks were carried out at intervals with solutions of pure urea, when theoretical recoveries were always obtained.

Feces were not usually tested, the fecal nitrogen generally being taken as one-ninth of that in the urine.

Exudate. The exudate was collected in Cellucotton or wool, the dressings being covered with washed,* sterilised Cellophane placed immediately under the crêpe pressure bandages to minimize leakage.** The nitrogen in the dressings was determined by boiling them with 10 per cent sulfuric acid and taking a sample of the mixture for Kjeldahl digestion. It was found necessary to keep the disintegrated dressings vigorously stirred in order to prevent bumping: this stirring has the further advantage of breaking up the dressings and assisting in the formation of a uniform mixture. A blank value for dressings and reagents was obtained by treating in the same way an approximately equal quantity of the materials employed. The results were further corrected for the nitrogen in the penicillin-sulfathiazole cream used, it being assumed, for purposes of calculation, that little or none of the sulfathiazole had been absorbed. (Absorbed sulfathiazole will have been excreted in the urine, so that the error introduced by this procedure in calculating the total balance is negligible, especially in view of the other sources of error. For accurate assessment of the exudate nitrogen some account would of course have to be taken of the exact amount of sulfathiazole remaining in the dressings.) Duplicate determinations on samples of the uniform mixture generally agreed to within 0.5–1 per cent.

Earlier estimations of exudate protein were made by Miss E. Semeonoff by soaking the dressings in water, treating a sample of the solution with sulphosalicylic acid and matching the resultant turbidity with the permanent standards of King and Haslewood.[4]

Food. A fraction, usually one fifth, of the food was kept aside and at the end of the balance period ground to a uniform paste. A portion of this (e.g. 50–100 Gm.) was heated on a water bath with a little sulphuric acid for several hours, and samples of the resulting solution or fine suspension were taken for Kjeldahl analysis, the digestion mixture of Chibnall, Rees and Williams[5] being used. In most of the balances eggs were not tested, on account of the food shortage, and the nitrogen was calculated from tables (McCance and Widdowson[6]) on the basis of the number and average weight of eggs consumed. All other nitrogenous foods were analysed.

* The Cellophane was washed in order to remove a water-soluble nitrogenous substance that might have contaminated the dressings. The washed film was dried and autoclaved before use.

** While every effort was made to avoid loss it is probable that in some cases there was a slight leakage of exudate.

Vomit was homogenised in the same way as food, and a portion taken for analysis.

Plasma protein was estimated by the micro-digestion and nesslerisation method of King.[7] Checks on reagents, digestion and colorimetric matching were carried out at intervals by using pure solutions of glycine, when satisfactory results were obtained. In some cases serum protein was estimated, with the phenol reagent of Folin and Ciocalteu (Greenberg, 1929[8]), in Mr. Garfield Thomas's laboratory.

Urinary creatinine and creatine were determined by Folin's[9] method. In a few cases a modified Benedict method for total creatinine, described by Macy,[10] was tried: it consists in evaporating the urine with normal hydrochloric acid, the drying being completed on a water bath to avoid charring and the lead treatment being omitted. Macy states that this method was found satisfactory in her study of creatine excretion in children; but she investigated normal children, and in the present writer's experience the residue from the urine of burned patients sometimes darkens sufficiently on evaporation with hydrochloric acid to give a high result. Creatinine and creatine estimations were usually done within 24 hours of receiving the urine.

Plasma chloride was determined on 0.5 or 1 ml. by digestion with silver nitrate in nitric acid and back titration with alcoholic potassium thiocyanate after the addition of alcohol. Blood was collected under liquid paraffin and the plasma separated as soon as possible.

Urine chloride was determined by the Volhard method, essentially as described by King.[7]

Blood sugar was estimated by the method of Folin and Wu adapted for 0.1 ml., described by Harrison.[11]

Calculation of the areas burned was done by the surgeon and was based on Berkow's[12] values.

RESULTS

Urinary nitrogen excretion. The urinary nitrogen excretion was measured in 20 cases, in 7 of which nitrogen balances were carried out. Results in a number of other cases are omitted for the reason given above. The cases are arranged in four arbitrary groups, as in the Glasgow Burns Unit report (L. Colebrook *et al.*, 1944[13]), viz.:

Group I: 1– 5 per cent of body surface burned
Group II: 6–15 per cent of body surface burned
Group III: 16–30 per cent of body surface burned
Group IV: over 30 per cent of body surface burned

The results are summarized in Table I, from which it will be seen that few of the urinary nitrogen figures in Groups I and II are excessive and that some are in fact low. The highest nitrogen output observed was in Case 15, a man of 28 with 20 per cent burns, who excreted 40 Gm. of nitrogen in a 27-hour collection of urine beginning three days after the burning. This high loss

TABLE I.—*Urinary Nitrogen Excretion in Burn Cases*

Case No.	Age, Sex	Burn Total	Burn Deep	Daily Urinary Nitrogen	Plasma Transfused	Remarks	Lived or died (day)
				GROUP I CASES			
1	54, F	5%	0	Average 8.0 Gm. N per day for first 8 days	None	Epileptic. Admitted with slight infection of burns (quickly controlled)	L.
2	25, M	2½%	1%	Average 10.1 Gm. N per day for first 3 days	None	Afebrile. Uninfected	L.
3	37, M	2½%	1½%	Average 13.8 Gm. N per day from 2nd to 5th day	None	Afebrile after 3rd day. Uninfected	L.
4	58, M	1%	1%	Average 14.5 Gm. N per day for first 6 days	None	Afebrile after 3rd day. Uninfected	L.
5	13, M	4%	0	Average 7.2 Gm. N per day from 2nd to 6th day	None	Febrile first week, but uninfected	L.
				GROUP II CASES			
6	10, M	6%	0	Average 4.9 Gm. N per day for first 13 days	None	Afebrile after 3rd day. Uninfected	L.
7	30, M	15%	5%	Average 15.6 Gm. N per day for first 18 days (no urine collections on 9th, 10th, 11th, 14th and 15th days)	1 litre	Slight pyrexia with low grade infection first 6 weeks	L.
				Ml. Urine (24 hrs.) Gm. N			
8	16, M	7%	3.5%	530 (2nd day) 8.2 2260 21.9 2730+ 16.1+ 2570 16.95 1685 17.2 2200 23.1 2020 21.8 1200 23.65 990 16.3 1280 19.7 990 13.3 1270 21.7 1000+ 13.7+ 1115 19.7	None	Clinically uninfected throughout, but slight pyrexia for first 7 days. (See case notes below)	L.
9	29, M	6%	0	1900 12.2 1930 16.6 1860 19.5 1750 20.6 2550 21.4 1700 19.0 1400 17.0 1575 12.1 1560 18.3 1700 13.8	None	Clinically uninfected throughout. Afebrile after first 48 hours. (See case notes below)	L.
10	9, M	6%	3%	Average ca 7 Gm. N per day from 2nd to 7th day	None	Low grade infection with pyrexia 2nd week	L.
				Ml. Urine Gm. N			
11	58, M	9%	4%	1070 (6 hrs) 4.0 1300 (13 hrs) 7.05 2480 (23 hrs) 15.4 390 (1 hr) 1.65 1455 (24 hrs) 9.25 2595 (26 hrs) 20.75 1610 (22 hrs) 10.2 2595 (24 hrs) 15.5 470 (1½ hrs) 1.35	None	Clinically uninfected and afebrile throughout. (See case notes below)	L.

TABLE I.—*Urinary Nitrogen Excretion in Burn Cases (Cont.)*

Case No.	Age, Sex	Burn Total	Burn Deep	Daily Urinary Nitrogen	Plasma Transfused	Remarks	Lived or died (day)
12	27, F	9%	7%	Ml. Urine (24 hrs.) Gm. N			
			10 days after admission	1100 (17½ hrs) 5.55	None	Clinically, slight low grade infection. Temperature never above 100° F. (See case notes below)	L.
				1840 9.8			
				1880 9.2			
				1010 6.15			
				1400 7.35			
				1850 10.85			
				....			
				900 (6 hrs) 6.05			
				365 (18½ hrs) 3.15			
				830 13.35			
				1385 8.6			
				730 13.6			
				1160 (27 hrs) 8.5			
				1280 8.25			
				1135 3.4			
				1400 5.3			
				1950 7.1			
				2010 7.65			
				1150 (22 hrs) 4.85			
				1975 8.5			
				2060 7.35			
				1820 (25 hrs) 6.95			
13	58, F	15%	10%	Urine + exudate loss given in Table 2	2.2 litres	Low grade fever (See case notes below)	D. (14)
				GROUP III CASES			
14	7, F	30%	30%	Average ca 6 Gms. N per day for first 9 days (but some specimens incomplete)	Nearly 2 litres	Burns uninfected but some pyrexia first few days	L.
15	28, M	20%	15%	Average 19.3 Gm. N per day for first 11 days (omitting 2 incomplete collections), and 15.7 Gms. per day for next 14 days (omitting 2 incomplete collections). Highest output was 40.1 Gm. in 27 hrs. 3–4 days after burning	5.6 litres	Low grade infection and little fever first 2 weeks	L.
				Ml. Urine (24 hrs.) Gm. N			
16	23, F	22%	17%	680 (4 hrs)	5.8 litres	Febrile first 8 days. (See case notes below)	D. (25)
				840 (16½ hrs) 3.25			
				1365 8.5			
				780 10.2			
				725 10.0			
				1505 13.2			
				2070 15.2			
				2105 17.4			
				2260 15.2			
				2310 17.0			
				2285 16.0			
				2610 18.45			

TABLE I.—*Urinary Nitrogen Excretion in Burn Cases (Cont.)*

Case No.	Age, Sex	Burn Total	Burn Deep	Daily Urinary Nitrogen	Plasma Transfused	Remarks	Lived or died (day)
				GROUP IV CASES			
17	21, F	72%	50%	Average 19.5 Gm. N per day for first 11 days (? some specimens incomplete)	7.6 litres	Some fever throughout. ? Chest infection	D. (12)
18	14, F	73%	58%	Average 10.7 Gm. N per day for first 7 days (omitting one incomplete collection), and 11.9 next 10 days. Then incontinent of urine	5.2 litres	Febrile throughout. Endocarditis and low grade infection of burns	D. (90)
				Ml. Urine (24 hrs.) Gm. N			
19	17, M	60%	25%	520 (18 hrs) 3.3 1500+ 7.2+ 1330 9.4 530 6.4	22 litres (approx.)	Febrile throughout. Pyocyaneus infection	D. (4½)
20	4, F	50%	50%	See nitrogen balance notes below	2.9 litres	Febrile 5 weeks with low grade infection. (See case notes below)	L.

of nitrogen in the urine was not maintained, however, and his average daily excretion over a number of days, though above the normal amount, was not as high as might be expected. Case 19, a boy of 17 with 60 per cent burns, excreted small amounts of nitrogen in the urine during the four and a half days he lived. Some kidney damage was found at autopsy and this was confirmed by histological examination. This patient received methionine by mouth two days after admission, but it is impossible to say what effect it had. Case 18, a girl of 14 with 73 per cent burns, was given methionine at the suggestion of Professor R. A. Peters; but as by that time the patient was incontinent of urine any effect the methionine might have had on the urinary nitrogen excretion was not apparent.

Nitrogen balances. Table II gives details of 16 nitrogen balances carried out in 7 cases of burns ranging from 6 to 50 per cent of the body surface. Detailed case notes are given below, and these are followed by a discussion of the findings. Unfortunately, it was impracticable to weigh any of the patients (except Case 20, a small child) as the necessary apparatus was unobtainable at the time.

In Table II a distinction is made between the nitrogen taken in the food and that of intravenous plasma, and where possible between urine nitrogen and exudate nitrogen. In the experience of Major J. A. F. Stevenson,[14] plasma given intravenously does not immediately increase the urine nitrogen, apparently being simply added to the pool of body protein (cf. Best and Taylor[15]). This means that in nitrogen balance experiments in burns there are two things to consider:

(a) the total nitrogen balance over a period, including any plasma protein lost in the exudate and protein given intravenously;

TABLE II.—*Summary of Nitrogen Balances in Terms of Average Daily N Intake and Output Over Each Balance Period.*

Case No., Sex, Age, Extent and Time of Burn	Balance Period	Average Daily N Intake (Gm.)			Average Daily N Loss (Gm.)			Nitrogen Balance (Gm./Day)	Remarks
		By Mouth	I.V. Plasma	Total	Urine + Faeces	Exudate	Total		
No. 8, M, 16	6 a.m. 10/8-10 a.m. 17/8	8.6	0	8.6	23.1+	?	23+	−14.5+	Large quantities of exudate
7% (3.5% deep)	10 a.m. 17/8-10 a.m. 22/8	20.0	0	20.0	19.2	.....	19.2	+ 0.8	Slight exudation
4 p.m., 9/8/45	10 a.m. 29/8-M.D. 3/9	23.3	0	23.3	14.7+	.....	14.7+	+ 8.6 ca.	Serum protein 9/8/45 6.4 Gm./100 ml. 18/8/45 5.6 Gm./100 ml.
No. 9, M, 29	M.N. 4-5/9-10 a.m. 10/9	24.6	0	24.6	18.5+	0.4	18.9+	+ 5.7−	Average calorie intake from 17-22/8/45, 2700 per diem; from
6% (superficial)	10 a.m. 10/9-10 a.m. 15/9	24.7	0	24.7	17.8	.....	17.8	+ 6.9 ca.	29/8-3/9/45, 3270 per diem
11.30 p.m., 4/9/45									5/9/45 Serum protein 5.9 Gm./100 ml.
									9/9/45 Serum protein 6.1 Gm./100 ml.
No. 11, M, 58	1 p.m. 14/6-9.30 a.m. 20/6	19.75	0	19.75	16.15	0.15±	16.3±	+ 3.45 ca.	Average calorie intake from 6-10/9/45, 3900 P.D.; from
9% (4% deep)									11-14/9/45, 3800 P.D.
10.30 a.m., 14/6/46									14/6/46 Plasma protein 6.4 Gm./100 ml.
					+ vomit				15/6/46 Plasma protein 6.1 Gm./100 ml.
No. 12, F, 27	3.30 p.m. 7/5-3 p.m. 14/5	14.7	0	14.7	10.3	0.5	10.8	+ 3.9	20/6/46 Plasma protein 6.1 Gm./100 ml.
9% (7% deep)	1.30 p.m. 15/5-11 a.m. 20/5	4.3	0	4.3	10.75*	1.0	11.75*	− 7.45 ca.	1 day's urine (13/5-14/5) thrown away. Probable N content
9.50 p.m., 27/4/46	11 a.m. 20/5-11 a.m. 25/5	10.6	0	10.6	7.25‡	?	7.25+	+ 3.35 ca.	allowed for. Grafted between 1st and 2nd balances
	11 a.m. 4/6-10 a.m. 8/6	12.85	0	12.85	7.70	.....	7.70	+ 5.15	* Some vomit not kept
									‡ Figure includes menstrual blood
No. 13, F, 58	1.25 p.m. 15/5-3 p.m. 22/5	6.9	2.5	9.4	Urine, exudate, vomit & faeces		10.4+	− 1.0+	Plasma protein 24/5/46 5.4 Gm./100 ml.
15% (10% deep)									Plasma protein 4/6/46 4.9 Gm./100 ml.
12.20 p.m., 15/5/46									Plasma protein 13/6/46 5.7 Gm./100 ml.
									(See Figure 1 for plasma protein levels)
No. 16, F, 23	3.30 p.m. 15/3-11 a.m. 21/3	8.25	7.95	16.2	11.3	3.5	14.8	+ 1.4 ca.	Until 17/3/46 patient had only sodium lactate by mouth.
22% (17% deep)	11 a.m. 21/3-2.30 p.m. 26/3	18.7	0	18.7	17.5	2.95	20.45	− 1.75	Caloric intake from 17-21/3/46 averaged 1200 per diem
10.30 a.m., 15/3/46									Caloric intake for 2nd period averaged ca 1650 per diem from milk, eggs and glucose (not counting bread, meat and potatoes)
									See Figure 1 for plasma protein levels.
No. 20, F, 4	6 p.m. 27/6-5 p.m. 4/7	2.9	3.3	6.2	Urine, exudate, faeces, vomit		8.05	− 1.85	Approx. Caloric Intake/Day. Weight
50% (Deep)									1st Period .... 4/7/46 47 lbs.
9.30 a.m., 27/6/46	5 p.m. 4/7-10 a.m. 11/7	7.8	0	7.8	Urine, exudate and faeces		6.6	+ 1.2	2 " 1500 15/7/46 45 lbs.
	17/7-21/7	10.5+	0	10.5+				?	3 " 1350+
	26/7-30/7	9.5	0	9.5				?	4 " 1090+ 25/7/46 47 lbs.
	5/8-8/8	12.0±	0	12.0±				?	5 " .... 8/8/46 39 lbs.
	M.D. 19/8-3 p.m. 24/8	16.4	0	16.4	8.3	0.6±	8.9±	+ 7.5 ca.	6 " 1450+ 28/8/46 42 lbs.

(b) the metabolic state of the patient on a particular day or over a period ("metabolic" nitrogen balance), in calculating which, according to Stevenson, intravenous plasma and protein lost in the exudate should probably not be included.

GROUP II CASES

Case 8.—B. S., a boy of 16, received flash burns of the face and hands while at work on 9/8/45 and was admitted on the same day. His serum protein, as determined colorimetrically with the phenol reagent, was 6.4 Gm. per 100 ml. on admission. A nitrogen balance was started next morning and carried on until the dressings were changed a week later, when a new balance was commenced. Unfortunately a satisfactory technic for the estimation of the nitrogen lost into the dressings had not at the time been evolved by us, and the negative balance was certainly larger than the figure given in the table (as indicated by the plus sign). Over the second balance period, which consisted of five days, the patient was more or less in nitrogen equilibrium (neglecting some slight exudation during this period), although nine days after admission his serum protein, determined as above, had fallen to 5.6 Gm. per 100 ml.; and in the third period he was in marked positive balance. The urinary nitrogen losses were somewhat above normal levels but the large negative nitrogen balance in the first period was evidently due chiefly to the small amount of food eaten during the first few days.

The patient was clinically uninfected throughout but had slight pyrexia for the first seven days. Bacteriology: chiefly Gram-negative cocci after the first three days, later staphylococci.

Case 9.—W. P., a man age 29, received blister burning of the right ankle joint and knee on 4/9/45 through coming into contact with boiling water. A nitrogen balance was commenced on admission at midnight, but unfortunately some urine was discarded by the night staff. However, the quantity lost is believed not to have been large, and urine collection was resumed at about 10 A.M. on the following morning. One breakfast was not sampled in the absence of the metabolic nurse and the approximate nitrogen content was calculated with the aid of tables. Serum protein, measured with the phenol reagent, was 5.9 Gm. per 100 ml. on 5/9/45 and 6.1 on 9/9/45, i.e., towards the end of the first balance period, during which the patient was well in positive nitrogen balance. A second balance experiment over the next five days showed him to be again in positive nitrogen balance. By 21/9/45 the patient was practically healed, and he was discharged on the 24th.

The patient was clinically uninfected throughout, and afebrile after the first 48 hours. Bacteriology of burned area: sterile for first 6 days, then a few micrococci.

Case 11.—J. B., a man of 58, burned his right hand and arm and right knee when he fell over a pot of molten metal on 14/6/46. A nitrogen balance was started soon after admission. The plasma protein was 6.4 Gm. per 100 ml. 4½ hours after burning and 6.1 on the next day. It was still 6.1 at the end of the balance period, over which the patient was in positive nitrogen balance. No more balance experiments were done in this case.

The patient was clinically uninfected and afebrile throughout, and healed in four weeks. Bacteriology of burned area: sterile for first six days, then diphtheroid bacilli and non-haemolytic streptococci (on one occasion only).

Case 12.—P. G., a woman of 27, an epileptic, had a fit on 27/4/46 and fell onto the fire. The patient was treated at another hospital before being transferred to this hospital on the following day. Nitrogen balances were started on 7/5/46. Skin grafting operations were carried out on the 16th: the adverse effect on nitrogen balance was due

to a general reduction in food intake, not to any significant increase in urine nitrogen. It should be noted that the figures given in Table 2 take no account of blood lost during grafting operations between balances. Subsequently it was not difficult to get the patient into positive nitrogen balance, though plasma proteins remained low for a time. Urinary nitrogen excretion also remained at a low level (see Table I).

Clinically there was a slight low grade infection by micrococci, non-haemolytic streptococci, coliform bacilli and staphylococci. The temperature was never above 100°F.

Case 13.—A. R., a woman of 58, also an epileptic, had a fit and fell onto the fire on 15/5/46. Plasma protein soon after admission was 6.9 and fell rapidly to 5.5 Gm. per 100 ml. 8½ hours later (Fig. 1). A nitrogen balance was started on admission and continued until the dressings were changed on 22/5/46. Owing to incontinence an uncertain amount of the urine was lost and some also leaked into the dressings together with a little feces, so that it is impossible to give an accurate figure for the nitrogen loss or to distinguish between urine and exudate nitrogen. It will be seen that the patient was in negative balance, though the urinary nitrogen averaged only about 8 Gm. per day. Plasma transfusions accounted for nearly 28 per cent of the total nitrogen intake during this period. Further balances were not attempted as the problem of collecting urine became too difficult. During changing of the dressings on 22/5/46 the patient collapsed with respiratory distress. Then the pulse, temperature and respiration rose and her condition remained critical. On 29/5/46 the plasma CO_2-combining power was found to be 40 volumes per cent and the plasma pH 7.30 (Mr. Garfield Thomas). Saline containing sodium bicarbonate and glucose was administered by Ryle's tube, but the patient died on the same day (29/5/46), probably as the result of a pulmonary embolism. A few days before death she developed a sudden transient right-sided hemiplegia.

The burned areas were clinically uninfected but there was low grade infection for the 14 days she survived. Bacteriology: staphylococci and Gram-negative cocci.

GROUP III CASE

Case 16.—J. R., a woman of 23, was subject to "dizzy spells" that had been diagnosed as epilepsy at some previous time. During one of these fits, on 15/3/46, she fell onto the fire and received burns mainly over the right side of the body—neck, pectoral region, scapular region, hip, buttock, arm and hand. A nitrogen balance was started soon after the burns had been dressed, and carried on until the next dressing, which was on 21/3/46, when a new balance was begun. It will be seen from the table that the patient was in negative metabolic balance over the first period but in slight positive total nitrogen balance when the transfusions and exudate loss are taken into account. The total nitrogen in the plasma transfused was slightly more than that lost in the exudate over both balance periods. The patient then became incontinent of urine and no further balances were done. Plasma protein values are given in Figure 1. After a skin grafting operation on 2/4/46 the patient had an epileptiform attack which was repeated every few minutes. These fits became less frequent by 7/4/46, but on the 8th they recurred and she suddenly collapsed, became cyanosed and died. A specimen of cerebrospinal fluid obtained by lumbar puncture immediately after death was found to be quite clear and to contain:

Sugar	97 mgm per 100 ml (Folin-Wu)
Protein	5 mgm per 100 ml (turbidimetric with sulphosalicylic acid)
Chloride (NaCl)	777 mgm per 100 ml
Urea	47 mgm per 100 ml
Globulin:	no reaction (ammonium sulphate ring test)

The patient was febrile for the first eight days (low grade infection by proteus, pyocyaneus and staphylococci).

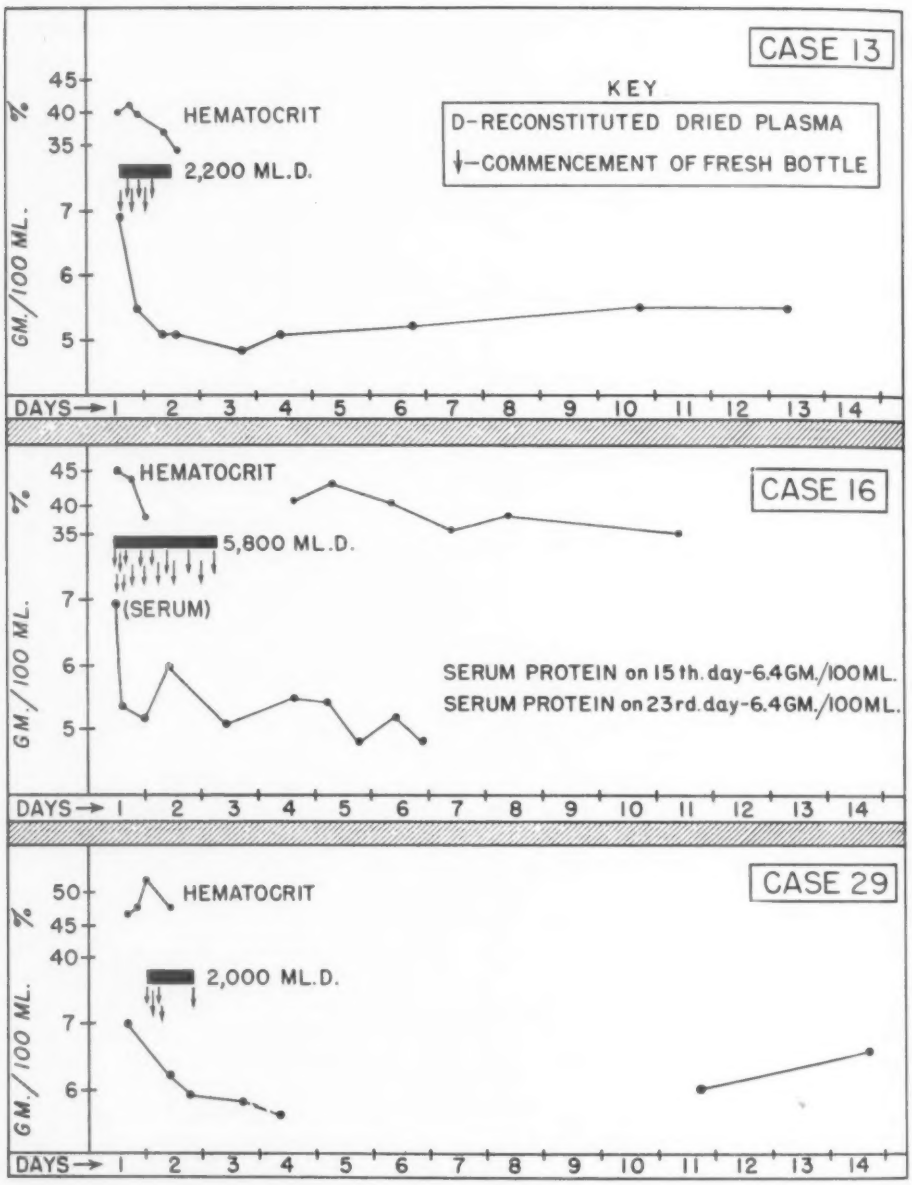

FIG. 1.—Plasma protein levels in three burn cases.

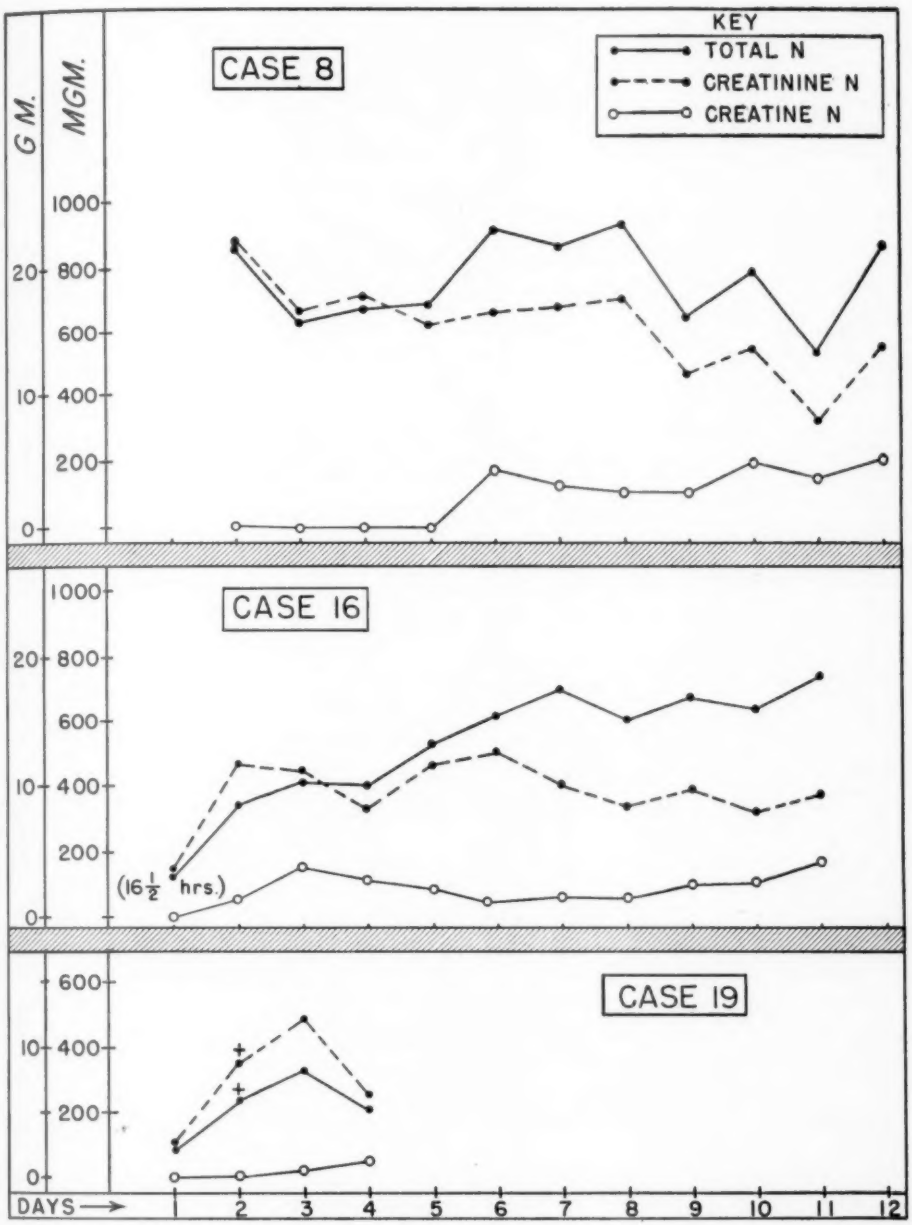

FIG. 2.—Urinary total nitrogen, creatinine N and creatine N excretion in three burn cases.

GROUP IV CASE

Case 20.—P. McN., a girl of 4, received severe burns at 9.30 A.M. on 27/6/46 when her small brother threw a lighted match at her, igniting her clothes. The burned area was estimated at 50 per cent (deep) and extended over trunk and arms. A nitrogen balance was started on admission and carried on until the dressings were changed 7 days later. After the third day the patient became incontinent of urine during the night, but it was found possible to collect the urine in Cellucotton. Unfortunately it was not possible to differentiate between urine and exudate nitrogen as the dressings became contaminated with urine and vomit. The patient was in negative balance over the first seven days and in slight positive balance over the second week. Third and fourth balances were commenced but the output could not be assessed on account of (a) complications arising from the necessity for only partially changing the dressings at different times, and (b) failure of the night staff to collect urine. However, a record was kept of the intake. A sixth balance was commenced on 19/8/46, by which time the patient was well on the way to recovery and in positive nitrogen balance. Skin grafting was done between the end of the second and beginning of the sixth balance periods. Owing to the extent of the burns it was not possible to do much blood analysis, but plasma proteins were estimated at intervals and were as follows:

29/6/46	6.2 Gm. per 100 ml
3/7/46	5.9 Gm. per 100 ml
8/7/46	6.0 Gm. per 100 ml
22/7/46	6.0 Gm. per 100 ml
27/7/46	6.2 Gm. per 100 ml
6/8/46	6.2 Gm. per 100 ml
16/8/46	5.8 Gm. per 100 ml
23/8/46	6.2 Gm. per 100 ml
6/9/46	6.8 Gm. per 100 ml

Records of the child's weight were kept and are given in the table. The loss of weight between the 25th July and 8th August followed a period of prolonged pyrexia—she was febrile for five weeks, with a low grade infection by staphylococci and Gram-negative cocci (later some coliforms).

Hemoglobin values (Haldane method) in this case were as follows:

3/7/46	52 per cent
8/7/46	74 per cent
17/7/46	64 per cent
24/7/46	87 per cent
27/7/46	57 per cent
8/8/46	66 per cent
10/8/46	94 per cent
16/8/46	86 per cent
23/8/46	84 per cent
29/8/46	90 per cent

Loss of blood during skin grafting procedures, and transfusion of packed cells, occurred between balance periods and are not included in Table II.

DISCUSSION

It appears that negative nitrogen balances in our cases were due to low intake rather than to a high output of nitrogen, and that when it was possible to get patients eating well it was not very difficult to get them into nitrogen

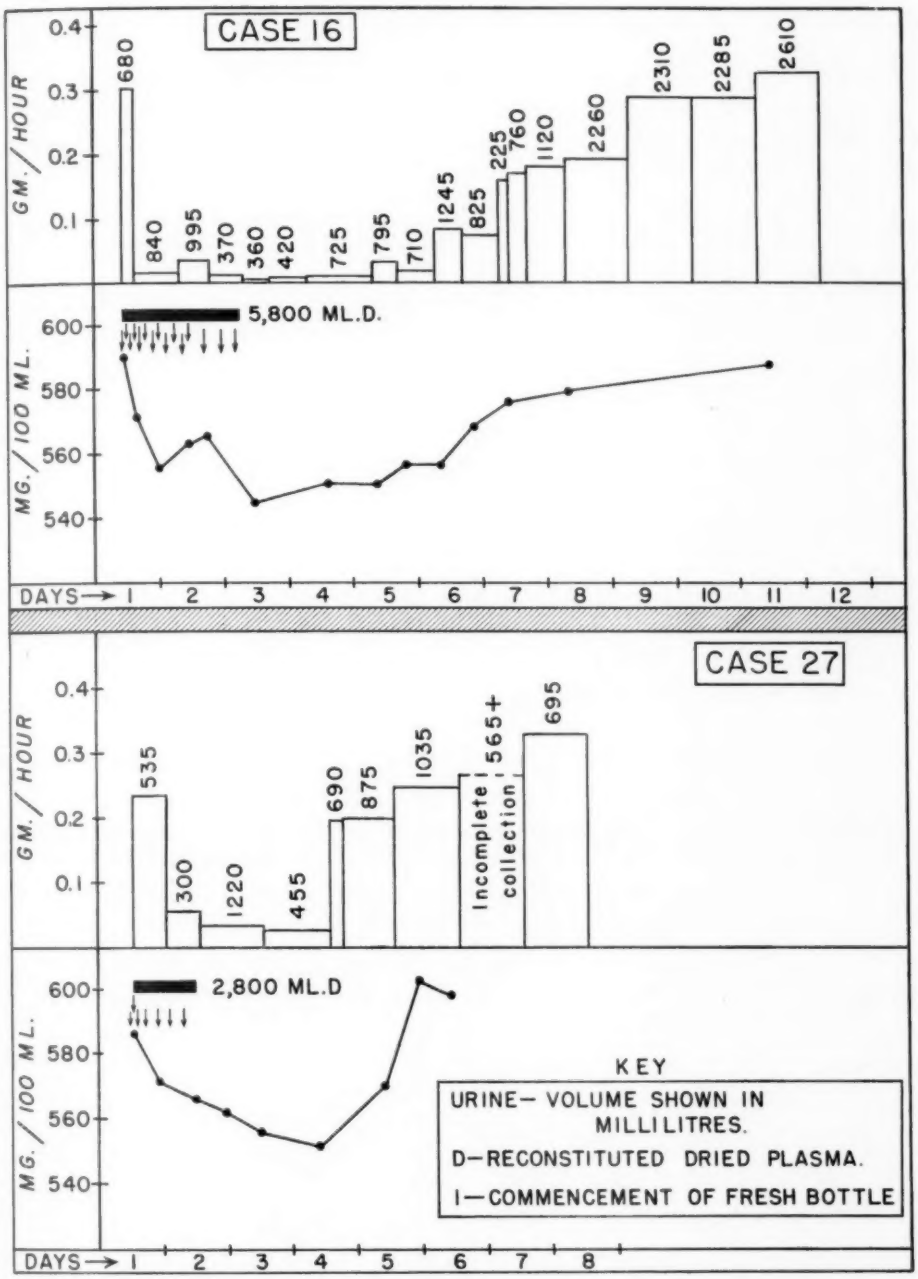

FIG. 3.—Urinary chloride excretion and plasma chloride levels.

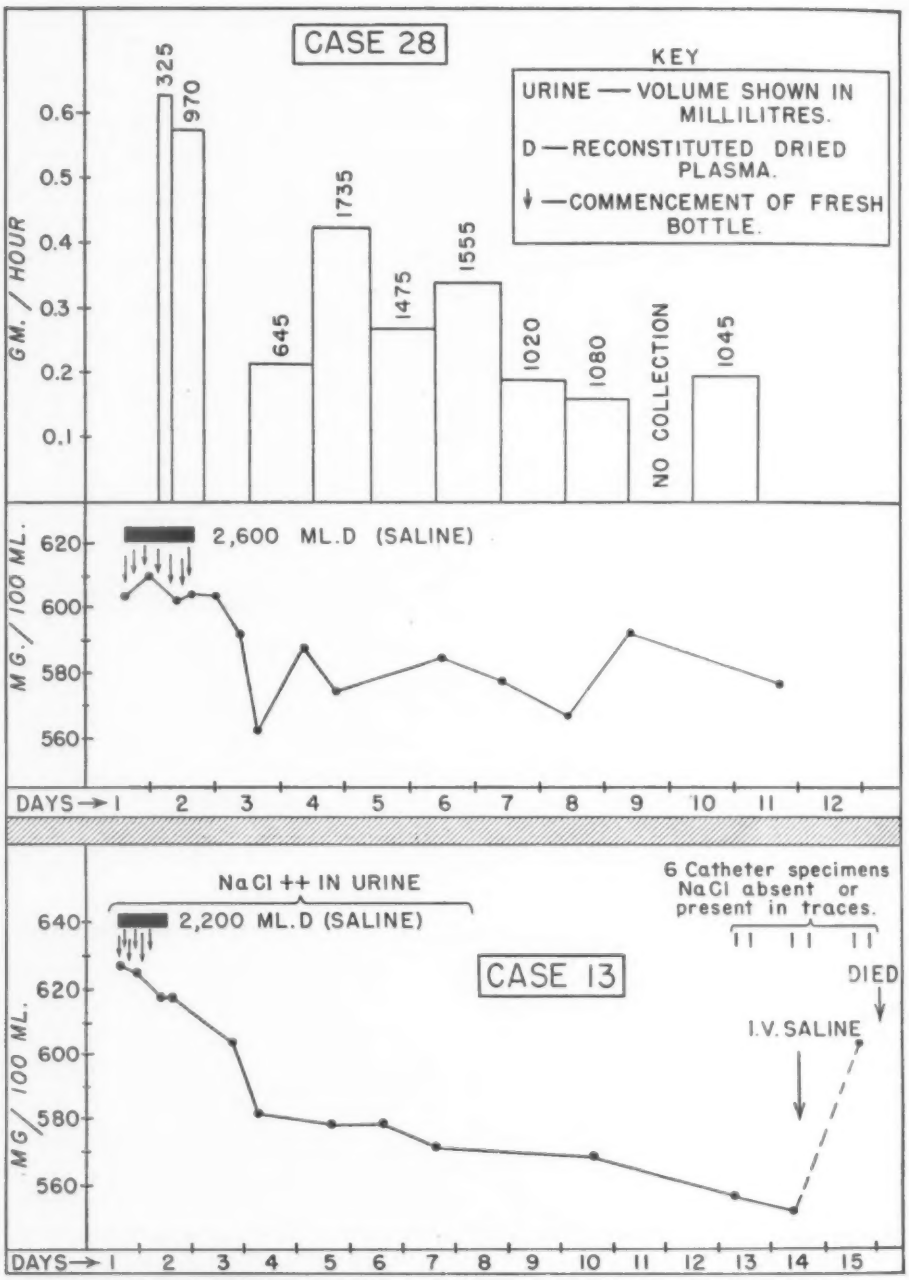

FIG. 4.—Urinary chloride excretion and plasma chloride levels.

equilibrium. Taylor *et al.*[16] and Co Tui *et al.*[17] have shown that there is a marked tendency for burned patients to get into negative nitrogen balance, and Co Tui has drawn attention to five possible sources of nitrogen loss: (1) intratissue loss into the burned areas; (2) loss in the exudate; (3) hemoglobinuria; (4) reduction in caloric and nitrogen intake as a result of anorexia; (5) the possible loss due to an "antianabolic period," probably as a result of altered hormonal balance. While it must be admitted that the loss in the exudate can be very large, we would draw attention to the fact that this loss, as well as the intratissue loss referred to in (1) above, is largely compensated (so far as total nitrogen balance is concerned) by the plasma transfusions. Proteinuria was encountered in most and hemoglobinuria in some of our severely burned patients, but the total amount of protein lost in this way was not large. With regard to the fifth source of loss, we have usually found the urinary nitrogen to be not markedly above the normal, and in fact some of our cases, especially women, excreted subnormal amounts of nitrogen in the urine. Our findings are in agreement with the statement of Cope *et al.*[18] that extensive deep burns are not necessarily accompanied by a large loss of nitrogen in the urine. Cope *et al.* attributed negative nitrogen balances in their cases to low caloric and nitrogen intakes, and the low excretion of nitrogen to the relative absence of infection. Most of our cases were relatively free from infection.

It seems likely that the nutritional state of the patient is at least as important as the extent of the injury in determining the level of nitrogen excretion, and it is possible that this may explain, to some extent, why we have not been able to confirm in all cases the large increases in urinary nitrogen output found by some of the American workers. Stevenson[19] refers to patients, previously in a poor nutritional state, who showed very little negative nitrogen balance after an injury; and Munro and Chalmers[20] have shown that the diet of the pre-fracture period has an important effect in modifying the metabolic response of rats to bone fracture.

Note on the food. Eggs and milk were used extensively and together contributed the greater part of the nitrogen and caloric intake of most of our balance cases. For example, in Case 20 (P. McN.) eggs accounted for from 22 to 36 per cent of the total nitrogen in each balance period, and milk 47 to 65 per cent. Casein hydrolysate was given orally in Case 12 (P. G.) but was poorly tolerated. It is doubtful whether hydrolysates are indicated except when digestion or absorption is impaired or when it is impossible to ingest sufficient protein in a more palatable form.

Supplements of glucose were given. Wherever possible the approximate caloric intake (calculated with the aid of tables) is shown in Table II.

It may be that protein and essential amino acids considerably in excess of the amounts adequate for maintenance of nitrogen balance are required for healing, but we have no exact data bearing on this.

NITROGEN IN THE EXUDATE FROM THE BURNED AREA

The nitrogen in the exudate was measured in seven other cases, in which

complete balances were not carried out, and the results are included in Table III together with those for Cases 9, 12 and 16. Wherever possible the urinary nitrogen is shown for comparison. The practice of measuring the amount of protein lost in each 24- or 48-hour period (see lower part of table) was discontinued on account of the possible risk of infection involved in frequent changing of the dressings.

Pressure dressings to minimize loss of exudate from the burned area were used by Cope and Rhinelander[21] and by Koch[22] in America, and investigated experimentally by Cameron *et al.*[23] in this country.* Mr. J. McK. Duncan of

TABLE III.—*Nitrogen in the Exudate from the Burned Area*

Case No.	Age, Sex	Burn	Period	Exudate N (Gm.)	Urine N (Gm.)	Exudate N as % of total (excluding faeces)
21 (S.G.)	37, F	5% (superficial)	First 7 days	2.1	.....	.....
			Next 7 days	0.45	.....	.....
22 (J.B.)	7, M	5% (superficial)	First 5 days	1.75	33.6+	.....
			Next 5 days	0.5	53.7+	.....
9 (W.P.)	29, M	6% (superficial)	First 5 days	2.0	90.3+	ca 2%
12 (P.G.)	27, F	9% (7% deep)	First 7 days	3.5	55.0	6%
			Next 5 days	5.0	47.2	9.6%
23 (L.H.)	27, F	15% (8% deep)	First 7 days	11.7	30.2+	.....
16 (J.R.)	23, F	22% (17% deep)	First 6 days	20.2	60.4	25.0
			Next 5 days	15.3	84.1	15.4

The above values for exudate N are calculated as described under "Methods," and are therefore likely to be slightly low (depending on how much of the sulfathiazole was absorbed).

2 (G.J.)	25, M	2½% (1% deep)	First 24 hrs.	0.29*	30.2	1.8
			Next 24 hrs.	0.16*		
			Next 24 hrs.	0.09*		
24 (A.J.)	31, F	4% (1% deep)	First 1½ days	1.20*	.....	.....
			Next 48 hrs.	1.68*	.....	.....
25 (H.D'A.)	52, F	7% (superficial)	First 24 hrs.	1.38*	.....	.....
			Next 24 hrs.	1.92*	.....	.....
			Next 24 hrs.	1.47*	.....	.....
26 (J.M.)	9, F	8% (3% deep)	First 24 hrs.	0.67*	29.2	7.0
			Next 24 hrs.	0.55*		
			Next 48 hrs.	0.96*		

* Protein determined turbidimetrically +6.25.

this Unit has used pressure dressings in an attempt to control exudation, but we have no data demonstrating their effectiveness.

A quantity of blister fluid from a female, H. B., with blister burns of

* Britain.

the right hand and forearm was analysed and the results were as follows:

Total nitrogen	0.56 Gm. per 100 ml
Total protein	3.4 Gm. per 100 ml
Albumin/globulin	1.6
Chloride (NaCl)	490 mg. per 100 ml

It should be noted that as the clot of fibrin had already separated, the figure for total protein will be slightly low and that for the albumin/globulin ratio slightly high. Total protein and albumin were estimated by King's[7] nesslerisation method for plasma, in which the separation of albumin and globulin is effected with sodium sulphite (Campbell and Hanna[24]).

In a second case, W. H., a male aged 54, blister fluid (after separation of fibrin) was found to contain 5.1 Gm. of protein per 100 ml., the albumin globulin ratio being 3.3. The plasma protein in this case was 6.7 Gm. per 100 ml. and the plasma albumin globulin ratio 1.9.

PLASMA PROTEINS

In two of the balance cases (Nos. 13 and 16) serial plasma protein determinations over several days were made and are shown in Figure 1. A less steep fall in the plasma proteins was found in Case 29 (Fig. 1): this man of 58 had burns involving the face, right arm and shoulder, left forearm and hands, and was not transfused. It is possible that the steep fall in plasma proteins was partly due to dilution with the transfused reconstituted plasma, which contains only five grams of protein per 100 ml. In other burn cases low values were found.

CREATININE AND CREATINE EXCRETION

The urinary excretion of creatinine and creatine as compared with total nitrogen is shown graphically in Cases 8, 16 and 19 (Fig. 2). Results in 15 other cases are not shown, for reasons already given, but it can be stated that the high output of nitrogen on the third to fourth day (27 hours) in Case 15 (20 per cent burn) was associated with a high output of creatinine (2.75 Gm.) and creatine (1.55 Gm.). Creatine was found in the urine of two other adult males, L. H. and B. S. (Case 8), with 15 per cent and 7 per cent burns, respectively, but was absent or present only in small amounts in that of four other adult males with burns of not more than 2½ per cent. In some cases of creatinuria, in females as well as in males, there appeared to be a peak at the second to the fourth day, but this may have been fortuitous. In Case 19 (60 per cent burn), in which the low excretion of nitrogen has already been remarked on, very little creatine was passed in the urine.

Creatinuria has been reported as occurring in normal healthy males (Hobson[25]), but other workers, in extended studies, have failed to confirm this (cf. Tierney and Peters[26]). In certain of our cases it would seem likely that the creatinuria was to some extent a result of the injury and associated

pyrexia. Clark *et al.*[27] and Croft and Peters[28] found that after a severe burn rats showed a sharp rise in creatine excretion. Cuthbertson *et al.*[29] in experiments on rats showed that fracture of the femur caused a definite loss of creatine in the urine that paralleled the curve of nitrogen excretion, although preformed creatinine remained relatively constant; and Cuthbertson[30] reported similar findings in a study of human fractures.

It has been shown that the ingestion of readily assimilable carbohydrate is associated with creatinuria (Haldi and Bachmann;[31] Hobson[25]). Our burn patients received quantities of a sodium lactate-fruit juice mixture by mouth, and in view of the intimate connection of lactic acid with carbohydrate metabolism the possibility that the creatinuria may have been due partly to the lactate-fruit juice mixture cannot be excluded.

TABLE IV.—*Proteinuria in Burn Cases*

Group	Total Number of Cases	Protein Free	Traces of Protein Only	Protein
I	18	7	*9	2
II	17	3	†10	4
III	8	0	3	5
IV	6	0	1	5
Totals	49	10	23	16

* 5 of these cases showed proteinuria for the first day only. One case showed it on the 3rd day only.

† 2 of these cases showed proteinuria for the first day only. 2 cases showed no proteinuria on the first day, and two others none on the first two days.

PROTEINURIA

"Twenty-four-hour" collections of urine in 49 cases were tested for protein (sulphosalicylic acid test) for the first few days or longer. The results (Table IV) are in agreement with those found in the Glasgow Burns Unit (Anderson and Semeonoff[32]): thus only two* out of 18 cases in Group I had more than traces of protein in the urine, whereas five of the six Group IV cases had protein present in more than traces. Casts and blood cells were often found in the urine of these severely burned patients. A strongly positive reaction for protein was given in Case 20, a girl of four with 50 per cent burns, by a 14-hour collection starting from eight hours after the burn. The centrifuged deposit from the urine was found to contain a few granular casts and pus cells, but the urinary protein decreased to a mere trace within about four days. This patient made a good recovery, in spite of the extensive burns.

In two cases the urinary protein was estimated by Miss E. Semeonoff by the turbidimetric method using sulphosalicylic acid and the permanent turbidity standards of King and Haslewood.[4] One of these, a girl of 14 with 73 per cent burns (Case 18), excreted on an average 0.24 Gm. of protein per

* One of these two cases had an exceptionally large amount of protein in the urine, although the burn was only a light one (less than 1 per cent). There was almost certainly another cause for the proteinuria in this case.

diem over the first ten days; and the other, a woman of 21 with 72 per cent burns (Case 17), excreted an average of 0.66 Gm. per diem also in the first ten days.

CHLORIDE EXCRETION AND PLASMA CHLORIDE LEVEL

A marked decrease in the urine chloride, amounting in some cases to almost complete suppression of chloride excretion, appears to have been of general occurrence in the moderate and severe cases, the output returning to normal in the course usually of a few days. A decrease in urine and plasma chlorides in burns was observed by Davidson in 1926[33] and by other workers, though not all have confirmed the lowering of plasma chloride (see Harkins[34]). Figures 3 and 4 correlate chloride excretion with plasma chloride in four cases. Observations in Cases 16, 27 and 28, together with others in which a less complete picture was obtained, are in agreement with Davidson's suggestion that the lowered chloride excretion in burns is due not primarily to kidney change but rather to a lowering of the plasma chloride to below the renal threshold level (normally 562 mgm. NaCl per 100 ml. plasma). This does not preclude the possibility of kidney damage being responsible in some cases—compare Cases 13 and 29 (below). In Case 13 intravenous saline was given from 12:30 P.M. on 28/5/46 and the plasma chloride was 604 mg. per 100 ml. at 3:30 P.M. on 29/5/46, yet a specimen of urine obtained by catheterization at 6 P.M. on 29/5/46 contained hardly any chloride. It appears that excretion of urine had almost ceased, and *post mortem* the bladder was found to be congested and to contain a very small volume of purulent fluid.

It is worth noting that reconstituted plasma,* prepared from citrated blood, is low in chloride content when made up with distilled water. (A sample analyzed was found to contain 395 mgm. per 100 ml.) In view of the findings of Rosenthal,[35] Fox[36] and others, it was considered preferable to reconstitute the dried plasma with saline. In several cases normal saline was used by the surgeon, and this appeared to have the effect of delaying, if not entirely preventing, the fall in plasma chloride. It might possibly be better to use 0.2 per cent saline: this would bring the sodium chloride content of the reconstituted plasma to within the normal physiological range.

Cases 13, 20, 28 and 29 received plasma reconstituted with normal saline. In Case 20 chloride excretion in the urine was not diminished, as judged by qualitative tests, but on account of the extent of the burns the plasma chloride changes could not be followed. In the last of these cases (male, age 58, with 16 per cent burns), sodium chloride excretion was somewhat low, averaging 3.4 Gm. per diem during the first four days, although the plasma chloride appeared to be normal most of this time (cf. McIver[37]). Sodium chloride excretion rose to normal by the 24th day, but further observations of plasma chloride could not be made owing to the development of thrombosis.

* Birmingham, England.

In severe burns the amounts of reconstituted plasma given intravenously are often very large, sometimes amounting to several times the blood plasma volume. Mr. Garfield Thomas has suggested that the presence of citrate and (if the plasma has been reconstituted with distilled water) the deficiency in chloride might affect certain of the liver function tests by altering the balance of electrolytes—a point to bear in mind when interpreting the results of these tests.

TABLE V.—*Blood Sugar after Burning.*

Case No.	Age and Sex	Burn	Time of Burn	Time of Blood Sugar Level	Remarks	Blood Sugar (mg.%)
30	74, F	10% (8% deep)	2.45 a.m.	4.30 or 6.30 a.m.(?)	Lactate No previous history of diabetes ascertainable	298
31	15, F	15% (10% deep)	7.30 a.m. 7/12/45	10.30 a.m. 7/12	Lactate	137
				4.30 p.m. 7/12		162
				10.0 p.m. 7/12		112
				10.0 a.m. 8/12		105
				10.0 p.m. 8/12		127
				2.30 p.m. 9/12		125
				10.0 p.m. 9/12		138
32*	73, F	25% (10% deep)	3.30 p.m. 1/11/45	10.0 a.m. 2/11	Lactate	290
				2.15 p.m. 2/11	Died	355
				10.10 am. 3/11	6.35 p.m. 3/11/45	290
15	28, M	20% (15% deep)	13/4/45. Admitted 12.30 p.m.	3.30 p.m. 13/4	Lactate	123
				9.30 a.m. 14/4		161
3	37, M	2½% (1½% deep)	3 p.m. 28/12/45	6.0 p.m. 28/12	Lactate	106
				10.0 p.m. 28/12		106
				12.45 p.m. 29/12		137
				10.45 p.m. 29/12		130
				8.0 p.m. 30/12		118
22	7, M	5%	1.15 p.m. 18/2	4.0 p.m. 18/2	Lactate	139
				12 mdnt. 18–19		95
				9.30 a.m. 19/2		100
33	25, F	40% (32% deep)	9.45 a.m. 21/1/46	a.m. 21/1		111
23	27, F	15% (8% deep)	5.30 p.m. 17/1/46	6.30 p.m. 17/1	No lactate till	152
				10.30 p.m. 17/1	8.15 p.m.	118
				9.30 a.m. 19/1		133
34	5, F	10% (2% deep)	12.45 p.m. 31/12/45	4.0 p.m. 31/12	Lactate	128
				6.0 p.m. 4/1		103
16	23, F	22% (17% deep)	10.30 a.m. 15/3	11.30 a.m. 15/3	Lactate from 12	156
				4.30 p.m. 15/3	midday	128
35	30, M	3%	8.30 a.m. 15/3	10.0 a.m. 15/3		110

* This patient received treatment with insulin. Whether she had diabetes before burning is not known. A catheter specimen of urine obtained at 12.30 p.m. on 3/11/45 showed:

Sugar ++; Acetone +; Protein +; Chloride: trace.

BLOOD SUGAR

This was determined on venous whole blood or plasma (obtained for other estimations), by the method of Folin and Wu adapted for 0.1 ml. Findings are given in the table. In only two cases was a definitely abnormal value found; but as venous blood was used, the capillary blood sugar in a few of the others would possibly have been slightly above normal. These patients were given quantities of a sodium lactate mixture to drink, as recommended

by Fox (1944)[36], but how far this, or the fruit juice in which it was made up, tended to raise the blood sugar is not known.

SUMMARY

1. The daily urinary nitrogen excretion in 20 burn cases has been measured. In only a few was there an average daily nitrogen excretion markedly greater than normal, and in many patients it was somewhat low.

2. Sixteen nitrogen balances in 7 cases have been carried out and the findings are discussed. In five of these cases the nitrogen in the exudate from the burned area was measured. The well marked tendency for burned patients to go into negative nitrogen balance is confirmed, but negative balances in our cases seemed to be due to low intake rather than to increased loss of nitrogen.

3. Exudate nitrogen, measured in 10 cases, made up from 2 to 25 per cent of the total nitrogen output (excluding faeces).

4. A marked fall in plasma protein was observed soon after burning in three cases in which serial determinations were made over several days. Low plasma protein values were encountered in other cases also.

5. Creatinuria was observed in three adult males with burns of 7 per cent, 15 per cent and 20 per cent of the body surface.

6. Proteinuria was observed in many cases, especially in those with the more extensive burns.

7. Plasma chloride was determined at intervals in a number of cases and correlated with urine chloride. The fall in plasma chloride noted by some earlier investigators was confirmed and is discussed in relation to intravenous plasma therapy.

8. Blood sugar levels (venous blood, Folin-Wu method) after burns of various degrees of severity were measured in 11 cases. In two cases there was an undoubted hyperglycemia, and in a few others values were perhaps slightly high.

ACKNOWLEDGMENTS

It is a pleasure to acknowledge the help received from the Director of the Unit, Dr. L. Colebrook, F.R.S., and from Mr. Garfield Thomas, Biochemist to the Queen Elizabeth Hospital, Birmingham. The bacteriological notes in this paper are by Doctor Colebrook. Thanks are also due to Dr. D. P. Cuthbertson, Prof. R. A. McCance and Prof. R. A. Peters, F.R.S., for their help and interest in this work. I wish also to thank the Burns Unit Surgeon, Mr. J. McK. Duncan, and Dr. N. A. Thomas, Dr. W. H. J. Butterfield and Dr. M. Causer for much help; Sister R. M. Selley and Miss L. Thrussell, the metabolic nurses; Dr. R. E. O. Williams and Dr. E. Topley for the notes on post-mortem examinations; Mrs. F. E. Lowe for some hemoglobin values; Prof. G. Haswell Wilson for the histological examination referred to on page —; Dr. E. M. Hickmans and Miss E. Finch of the Children's Hospital, Birmingham, for advice on certain estimations; and Dr. J. R. Squire for reading the manuscript at an early stage. Acknowledg-

ments are also due to Miss E. Semeonoff, who did all the early biochemical work of this Unit until March, 1945.

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THE SURGICAL TRIANGLES OF THE INGUINOPECTINEAL REGION (INGUINA): THEIR CLASSIFICATION, PARIETAL RELATIONSHP AND SIGNIFICANCE IN HERNIA REPAIR

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FROM THE SURGICAL SERVICE OF THE VETERANS ADMINISTRATION

A CONCEPT HAS NOT BEEN PREVIOUSLY PROPOSED, in so far as we have been able to ascertain, which considers the inguinal region as a series of closely interrelated triangles approaching a composite pyramid.

Contributing to the existing confusion and consequent lack of accurate comparative criteria for surgical procedures in this area are: lack of an accepted classification of the surgical triangles of the inguinopectineal region, inaccuracy of anatomic description, variations and distortions of anatomic structures, failure to evaluate properly factors concerned in the development of hernia, faulty terminology and lack of a logical system of nomenclature.

Triangulation of the hernia-bearing region of the lower abdomen began in 1806 when Hesselbach[1] published a treatise on the origin of inguinal ruptures. He described the triangle medial to the inferior epigastric vessels with the sheath of the rectus and inguinal ligament forming its sides. Moreover, he made the original observation that it is the most vulnerable portion of the inguinal canal. The triangular concept of Hesselbach seemed to wane until Ferguson[2] (1895) recognized the frequent existence of a definite weakness in the medial angle of the canal. Since that time perpetual controversy has been going on over the minor ligaments and varying condensations of the fascia transversalis in or adjacent to this vulnerable triangle. These ill-defined fascial bands, such as Hesselbach's or interfoveolar ligament, internal crus of the internal ring (Browne[3]), Henle's ligament, the iliopubic tract, the ligamentum inguinale reflexum (Colle's fascia) and the femoral ligament are, for all practical purposes, anatomic frills. Since these fascial structures are inconstant and rarely of clinical significance in offering any structural support in reparative maneuvers, it would seem preferable to place less emphasis on them as definitive ligamentous or fascial entities.

An important clinical feature of the inguinopectineal region is its predilection to the formation of hernia. Contributing to this weakness are several predisposing factors: (1) the presence of so many orifices in the parietes which are essential for the passage of various anatomic structures. Although they are provided with fortifying fibers architecturally arranged for greater security, there inevitably exists a potential weakness at these points of vascular, visceral or urogenital exit. (2) Because of its dependency and the

concentric narrowing of the lower abdomen, the intra-abdominal pressure is increased in this region. (3) And most important of all, the anomalies which occur in the development of the undifferentiated abdominal wall plate concomitant with the descent of the testis. These various generic factors, anatomic, embryologic and dynamic, frequently coexist and collaborate in the evolutional process of herniation.

The whole hernia-producing region of the lower ventral abdominal wall is referred to collectively as the inguina. It is pyramidal in outline and on viewing it anteriorly and from within outward, it has depth or a third dimension. Such an observational view directs attention to the incipient development of hernia at its initial exit or fovea on the serous side of the abdominal wall. In contrast, it is difficult to reconstruct the retrogressive course of the hernial protrusion in the presence of a full-blown hernia after there has occurred structural distortion and parietal evagination. Early operations for the radical cure of hernia were focused exclusively on maneuvers for the eradication of the large saccular anomaly without giving adequate consideration to the role of the adjacent parietal structures. Lack of correlation and utilization of the parietal strata in the early reparative procedures resulted in anatomically imbalanced operations which temporarily delayed surgical progress in herniology. This technical gap is recognizable and is reflected in the evolution of some of the modern operative procedures.

Realizing the existing confusion and need for anatomic research in the inguinohypogastric region, Anson[4] and his associates, McVay,[5] Morgan[6] and Ashley[7] have made painstaking dissections of this region but confined their investigations chiefly to the transition of the musculofascial planes and the correlation of congenital and acquired anomalies. Much of our modern anatomic knowledge of this region stems from this study; unfortunately, however, the fascial and ligamentous boundaries of the controversial triangles were only casually mentioned by them. The need for integration of the surgical triangles and a better understanding of their relationship and variability to the hernial protrusion still exists. We should therefore like to present an anatomic outline of the triangles of the inguinopectineal region (inguina) that is applicable to the whole vulnerable or hernia-producing region. It is self-evident that if the surgeon is fully acquainted with the composite anatomy and the evolutional development of hernia, he should be better able to recognize and adequately to correct the faulty saccular or parietal weakness which predisposes to the formation of the hernia.

CLASSIFICATION OF THE SURGICAL TRIANGLES

Any given triangle should have a specific and unvarying anatomic boundary. Temporary alteration in size with co-existing distortion of its boundary by a herniated mass should not change the basic anatomic unit.

The classical anatomic arrangement as presented by the modern textbooks of anatomy and surgery is that of two distinct but, for the most part, unrelated inguinal and femoral regions. The region superior to the inguinal

ligament is known as the inguinal region and is the region intimately related to the development and repair of inguinal hernia. The region lying inferiorly

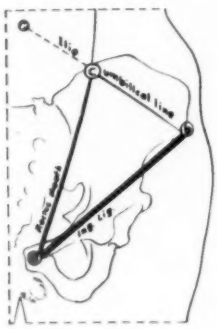


Fig. 1 Major superior triangle

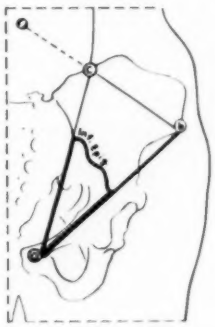


Fig. 2 Intermediate triangle (Hesselbach's)

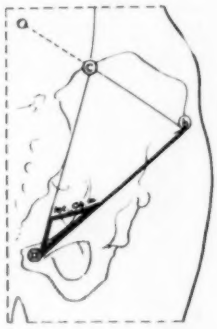


Fig. 3 Medial triangle

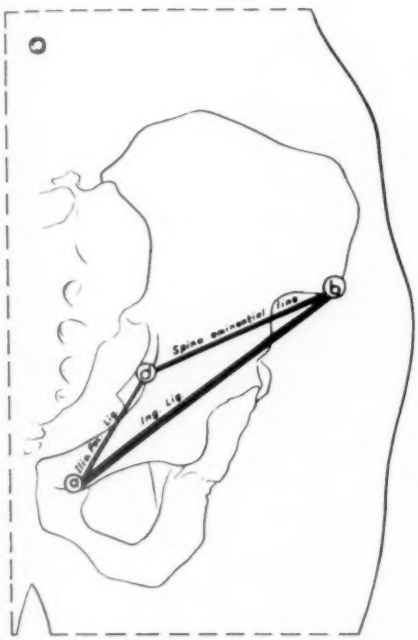


Fig. 4 Major inferior triangle

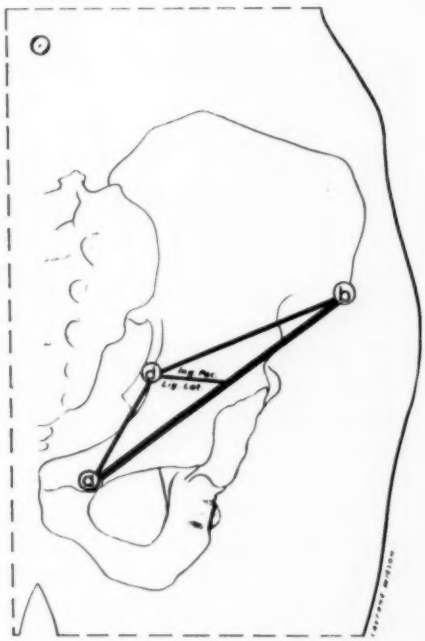


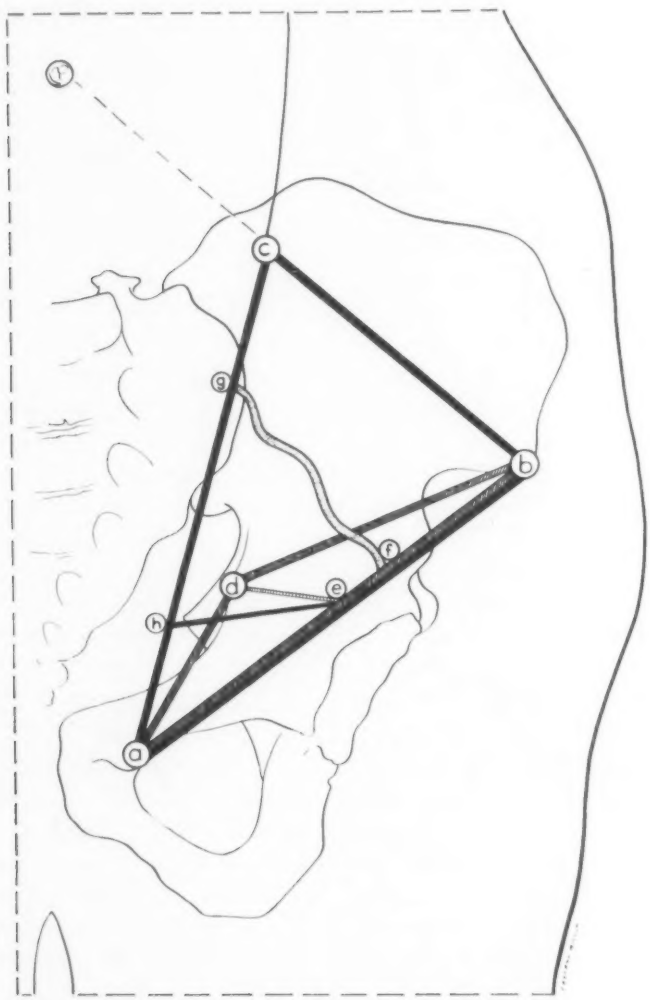
Fig. 5 Vascular and muscular triangles

a Pubic tubercle
b Anterior superior spine
c Junction, iliaumbilical line and rectus sheath
d Iliopectineal eminence

PLATE I.

and medial to the inguinal ligament is concerned with the development and treatment of femoral loculations of the sac and received little surgical con-

sideration until the publications of Moschcowitz,[8] Seelig and Tuholske,[9] Payne,[10] Dickson[11] and Wilmoth.[12] The recognition and correlation of these superior and inferior major triangular units in the repair of coexisting inguinal and femoral hernias, can hardly be overemphasized. Formerly,



a·b·c MAJOR SUPERIOR TRIANGLE
a·f·g Intermediate triangle
a·e·h Medial triangle

a·b·d MAJOR INFERIOR TRIANGLE
a·e·d Vascular triangle
b·d·e Muscular triangle

PLATE II.

when saccular ligation at varying levels of the funicular canal was the extent of surgical procedure and when there was still no recognized plan of repair of the canal, triangular integration was of less strategic importance. Therefore, in keeping with the proposed anatomic and surgical concept of the

inguinal region, a new classification of the triangles is described and illustrated which more nearly meets the basic anatomic principles upon which the various operative procedures may be founded.

Anatomically, the inguinopectineal region or inguina should be considered as one large pyramidal area, which is divided by a common base, the inguinal ligament into an extrapelvic or major superior inguinal triangle and an intrapelvic or major inferior inguinal triangle.

THE MAJOR SUPERIOR INGUINAL TRIANGLE (inguino-abdominal, inguino-hypogastric or inguinal trigone) (Plate I, Fig. 1) refers to that area of the lower ventral abdominal wall bounded medially by the lateral margin of the rectus sheath, inferiorly by the inguinal ligament and superiorly by that portion of the ilio-umbilical line which is lateral to the rectus sheath. The superior inguinal triangle contains (a) the intermediate or Hesselbach's triangle (Plate I, Fig. 2) which is the best known and first triangle to be described and (b) a smaller medial triangle (Plate I, Fig. 3). It is through the latter that incipient direct hernias are first manifest. It will be noted that these three surgical triangles have common medial and inferior boundaries, the rectus sheath and inguinal ligament respectively, but their superior boundaries differ. The inferior epigastric vessels limit the intermediate triangle and the inferior margin of the abdominal component of the internal oblique muscle the medial triangle. Actually, the medial triangle occupies the cleft between the abdominal and cremasteric portions of the internal oblique muscle. This vulnerable angle, which is devoid of overlying muscle fascicles to augment the fascia transversalis, was recognized by Ferguson, who, before the turn of the century, devised means of overcoming this mural weakness. More recently (1934) Andrews and Bissell[13] again directed attention to the importance of reinforcing this potentially weak area of the floor, which they designated the inguinal triangle, unfortunately adding to the already pre-existing confusion in the nomenclature of this anatomical region. Although the medial triangle is the smallest and is inconstant, it is unique in that it represents the pivotal area of weakness in the floor, marking the site of exit of all incipient direct hernias. This important anatomic fact and the need for its fascial reinforcement have not received the attention they deserve. Most textbooks of surgery emphasize or portray the site of emergence of internal or direct hernia as the intermediate or Hesselbach's triangle. Actually, on careful observation the initial exit of a direct hernia is in the medial triangle. This divergence of opinion is perhaps attributable to the frequent lack of opportunity to observe diminutive direct hernias at operation.

Most direct hernias when seen at operation are advanced and frequently occupy all of Hesselbach's space. At times, the protrusion may even displace the epigastric vessels laterally until they are at the level of the abdominal inguinal ring. In this case, a large direct sac obtains and the entire floor of the canal has been enfeebled by the pistoning action of the hernial mass. Coincident with this expanding hernia there is progressive enlargement of

the medial triangle until it is converted into and occupies the space normally occupied by the intermediate triangle.

Conversely, an enlarging indirect sac may displace the inferior epigastric

Fig. 1 Iliopectineal ligament (1)

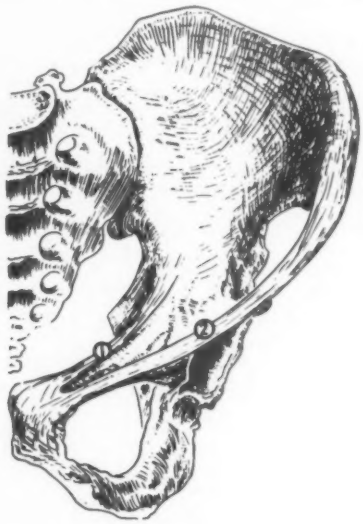


Fig. 2 Inguinal ligament added (2)

Fig. 3 Inguino pectineal ligaments medialis (3) and lateralis (4) added

Fig. 4 Composite of preceding ligaments, medial view

PLATE III.

vessels medially until they come to lie under the rectus sheath. In this instance, there no longer exists either an intermediate or medial triangular

space. However, despite these rare maximal distortional changes in the musculofascial structures, confusion should not exist and there should be no controversy over the triangular units if the normal anatomical boundaries are kept clearly in mind.

The major inferior triangle (Plate I, Fig. 4) comprises that space posterior and medial to the inguinal ligament and anterior to the bony pelvis. Its base or anterior boundary is the inguinal ligament (Plate III, Fig. 2) which is frequently referred to as the femoral or subinguinal region. The spinoeminential line which extends from the anterior-superior iliac spine to the pectineal eminence constitutes the posterior-superior boundary, and the iliopectineal line the posterior-inferior boundary of the triangle. This large inferior triangle is subdivided by the ligamentum inguinopectineal lateralis (Plate III, Fig. 3 and 4) into two smaller ones, the vascular and the muscular (Plate I, Fig. 5).

The vascular triangle is of much greater importance surgically. It occupies the interligamentous space or that area between the inguinal and iliopectineal ligaments. It is the more vulnerable triangle and represents the site through which the various types of femoral sacculations become manifest. Laterally, this triangle is limited by the ligamentum inguinopectineal lateralis which also separates it from the muscular triangle. Incidentally, this ligament is referred to as the iliopectineal in Callander's and Morse's textbook of anatomy, but actually it does not arise from the ilium superiorly but from the inguinal ligament and therefore it should be called the inguino-pectineal ligament. Similarly, the smaller ligament spanning the gap between the inguinal and iliopectineal (Cooper's) ligaments medially should be designated ligamentum inguinopectineal medialis (Plate III, Fig. 3 and 4) instead of lacunar or Gimbernat's. An even more glaring confusion exists in regard to the naming of Cooper's ligament (Plate III, Fig. 1) which extends from the iliopectineal eminence along the pecten of the superior pubic ramus to the spine of the pubis and is variously named pubic, superior pubic, pectineal and iliopectineal. Without entering into the historical controversy concerning Cooper's ligament, it is our contention that iliopectineal is the more descriptive term and the preferable designation, since it overlies and is intimately adherent to the uncontested iliopectineal line.

The muscular triangle is bounded anteriorly by the superior half of the inguinal ligament and posteriorly by the spinoeminential line. This line is depicted as straight on the drawings but is somewhat wavy in its proximal portion, corresponding to the anterior edge of the ilium. The inferior boundary of this triangle is formed by the ligamentum inguinopectineal lateralis. There are no normal apertures or potentially weak areas in this triangle as it is well fortified muscularly and herniations through it are exceedingly rare. It is included in the composite anatomic unit because occasionally pedicled fascial grafts are transferred through this triangular space to replace the inguinal ligament or to reinforce maximal attenuation of the fascia transversalis. By referring to Plate II it will be noted that the

muscular triangle differs from the other triangles of the inguina in that its apex is at the anterior-superior spine of the ilium, whereas the apices of the other five triangles (major superior inguinal with its component middle and medial triangles and the major inferior with its component vascular triangle) are at the pubic spine and all of these triangles have a common base, THE INGUINAL LIGAMENT.

THE PARIETAL WALL

The paries of the superior inguinal triangle (inguinohypogastric region) with the exception of slight fascial variants do not differ from the stratification of the lateral abdominal regions which is composed of three musculoaponeurotic layers. Each muscle stratum is enveloped by fasciae which fuse with the tendinous or aponeurotic plate lateral to their conjunction with the rectus sheath. Inferiorly the respective fasciae continue their descent into the scrotum, contributing the three strata of the funicular tube or canal. Generally, the transparietal portion of the canal is spoken of as the inguinal canal, whereas that portion caudal to the external ring is referred to as the extra-parietal or scrotal portion of the funicular canal.

The external or most superficial stratum of the abdominal wall is the external oblique muscle. The fascicular and tendinous portions of this muscle are the most constant in their proportions. Its broad aponeurosis stretches downward and is inferomesial to the anterior-superior iliac spine. In its inguinal portion is a triangular rent forming the external inguinal ring for the passage of the cord. The aponeurotic fault between the divergent crura is reinforced with arching membranous strands of connective tissue which arise from the fused epimysium and investing fasciae. The conjunction of the external oblique aponeurosis with the rectus sheath is medialmost of the three abdominal layers. The anterior and posterior enveloping fasciae of this muscle extend inferiorly and conjoin to form the external spermatic fascia. Laterally, the aponeurosis of this muscle is transformed into the inguinal ligament. The reflection of the lowermost fibers of the inguinal ligament at the inguinopectineal junction is usually demonstrable as thin, arching strands of connective tissue overlying the medial portion of the internal oblique muscle and are variously referred to as the triangular fascia, Colle's fascia, or ligamentum inguinale reflexum. This ligament makes an excellent anchorage for the first suture in the closure of the medial angle of the floor. Bisgard[14] has especially stressed the importance of this ligament in reinforcing the vulnerable angle of the canal. The length, constancy and uniform density of the external aponeurosis have made it a frequent contributor of single and occasionally multiple pedicled sutures, notably in the McArthur,[15] Robins,[16] Sachs,[17] Carscadden[18] and Joyce[19] technics.

The internal oblique muscle and its ensheathing fasciae constitute the second or intermediate layer. While there may be considerable differences in its fascicular and aponeurotic components, usually its medial third is tendinous. A frequent variation is its point of insertion onto the rectus

sheath at a variable distance from the pubic tubercle. This anomaly produces a triangular gap between the abdominal and cremasteric portions of the muscle. This vulnerable area in the floor of the canal was recognized by Ferguson, Halsted,[20] Bloodgood[21] and Andrews as a frequent predisposing factor in the formation of direct hernia. The older anatomies and surgical literature refer to the conjoined tendon, meaning the union of the aponeuroses of the internal oblique and transversus muscles with resultant formation of a common aponeurotic plate; however, currently this concept of aponeurotic fusion is not accepted by many anatomists (Anson, Morgan, McVay and Ashley). Suffice it to say, there has been sufficient anatomic research in recent years to support the view that the aponeuroses of the middle and inner strata actually do not conjoin, but in spite of this factual evidence the term "conjoined tendon" has been so long in use to connote a definite structure that it is not likely to be abandoned soon. An underlying accessory internal oblique muscle has been described by Tuholske and McVay and which occurs with sufficient frequency that to avoid possible confusion with the transversus abdominis its existence should always be suspected. The tendinous component of the internal oblique blends with the rectus sheath lateral to that of the external oblique muscle. Inferiorly, the enveloping fasciae of this muscle unite to form the cremasteric or middle layer of spermatic fascia.

The transversus abdominis muscle is the most deeply placed of the three parietal layers of the superior inguinal (inguinohypogastric) region. Owing to the variability of its fascicular, aponeurotic and fascial components, it has been and still is a perpetual source of controversy among anatomists. Naturally, this anatomic disagreement has resulted in a lack of unanimity as regards its surgical significance in the treatment of hernia. The muscle fascicles almost never extend inferiorly beyond the level of the abdominal inguinal ring and at this level they may be tendinous. Moreover, its transition from muscle to aponeurosis to fascia may be gradual or abrupt and may vary in density. Where there is arching of the aponeurosis, particularly if there is concomitant thickening, it is frequently referred to as the aponeurotic inguinal falx or conjoined tendon. The investment fascial coverings of this muscle do not differ from that of the other flat muscles.

The fascia transversalis is subadjacent and contiguous to the deeper stratum of the investing fascia of the transversus abdominis. It is currently interpreted as a distinct fascial lamina lining the inner surface of the abdomen. Almost a century and a half ago the anterior fascia was described and illustrated by Cooper in 1804 and in a subsequent edition of his book is referred to as the fascia transversalis. Zieman[22] considers this fascia as an intrinsic investing fascia of the abdominal wall and lining the abdominal cavity including the pelvis and the caudal surface of the diaphragm. A similar interpretation is implied by Dickson, who introduced the term endo-abdominal fascia. Similarly, Browne has given the same fascial lamina the name of abdominal connective tissue. The older anatomies (Shaw, 1825) refer to this fascial stratum as fascia longitudinalis, or reflexa of M. Cloquet.

The meticulous dissections of Anson have corroborated the broader concept of the fascia transversalis. Despite the varying evolutional interpretations of this fascial layer, from a surgical standpoint it should be considered the deepest fascial layer of the abdominal wall which is intimately related to the internal fascial bed of the transversus abdominis. These contiguous fascial layers are employed surgically as a single fascial plate. In the inguinal canal this internal parietal stratum is devoid of muscle fascicles caudal to the internal abdominal ring but possesses pivotal reparative value unless it has become attenuated by the hernial mass. The fascia transversalis continues laterally bridging the interligamentous space, and posteriorly encasing the femoral vessels, contributing to the reinforcement of the femoral ring before its insertion into the iliopectineal (Cooper's) ligament. With its extension caudally it becomes the internal spermatic fascia. In its medial course it splits, contributing to the formation of the anterior rectus sheath and the posterior rectus fascia. The latter should not be confused with the posterior rectus sheath. The transversus aponeurosis in its approach to the rectus muscle changes from bilaminar to unilaminar stratum at the semicircular line about 8 cm from the symphysis pubis. Inferior to this transitional line there is no posterior sheath, only a membranous film of rectus fascia. In the inferior segmental zone the aponeurotic fibers conjoin and run exclusively anteriorly to the rectus muscle, blending with the rectus sheath.

The difference of opinion regarding the reparative value of the mural strata has invariably centered on the innermost layer, the transversus aponeurosis and fascia from which arise so many ligamentous or equivocal fascial variants. The dividing structure between direct and indirect herma has long been accepted as the inferior epigastric vessels. Browne has challenged the strategic importance of these vessels and has pointed out that unsupported blood vessels have not the tenseness and rigidity which would enable them to act in this way. It is his contention that the condensation of fibers of the fascia transversalis which accompanies the vessels resists the progression of the pantaloon sac and separates the two major types of sac. It is the same band of connective tissue described by Hesselbach and is occasionally referred to as the internal crus of the internal abdominal ring. Some textbooks have named this fascial variant the interfoveolar ligament and ascribe its source to aberrant strands of muscle fascicles which have strayed from the transversus abdominis. Quain[23] shows an apparently identical structural departure superficial to the tissues bounding the internal ring and floor of the inguinal canal, but superimposes additional confusion by illustrating these tissues as continuous with the transversus aponeurosis and calling them the iliopubic tract. The anatomic dissections of Clark and Hashimoto[24] have corroborated the existence of the iliopubic tract and have revived Henle's ligament. They claim the latter arises from the lowermost portion of the rectus sheath and adjacent portion of the transversus aponeurosis. Inferomesially it blends with the iliopubic tract, a tough fibrous structure which is below and separate from the inguinal ligament. Briefly,

it would seem that while these fascial variants may be demonstrable, they are inconstant and to ascribe to them a strategic role in the fascial repair of the canal is exaggerating their significance. The error lies in overemphasizing aberrant fascial planes to the exclusion of the more important reparative problem comprising the whole floor of the canal and forgetting there are many predisposing anatomic factors in the genesis of initial and recurrent hernias.

COMMENT

Although basic but not wholly new, the unorthodox triangulation and proposed anatomical concept of an extra-pelvic and an intra-pelvic inguinal region would gain naught unless transposed into practical surgery. In considering reparative surgery of this region it is essential to redefine the loosely used term "defect" as contrasted with "weakness" if there is to be a composite understanding of the criteria and the reparative maneuvers employed in the fascialization of the floor of the canal. A defect in the wall conveys the erroneous idea that there exists a concentric aperture in an otherwise architecturally normal wall, a presumption which is rarely seen at operation except in the presence of interfascicular rents. Similarly, weakness may imply laxity of the wall or it may connote marked stretching with thinning of the fascia transversalis and internal oblique fibers until there no longer exists a homogeneous structure of strategic value. Where the latter obtains and the floor is membranous, its replacement is essential to the creation of an unyielding wall.

Assuming there is weakness of the entire extent of the canal with coexisting saccular protrusions, the strategy should be to form a resistant fascial diaphragm for the entire floor which in some instances is augmented by anchoring the cranial margin of the fascia transversalis to the iliopectineal ligament. Herein lies the advantage of a thorough understanding of the composite anatomy of the inguina.

The horizon of hernia repair has been decidedly increased by the proposed concept of triangulation and by the adoption of the superior inguinal approach for all hernias in the intrapelvic and extrapelvic triangles. This approach commands a more comprehensive view of the important ligaments and vessels, gives greater access to the intrapelvic hernia-bearing area and facilitates exposure of the iliopectineal ligament, which, if used for pivotal anchorage, achieves a more deeply placed parietal barricade.

CONCLUSIONS

The advantages that may accrue from a uniform and acceptable terminology of the mural and ligamentous structures of the inguinopectineal region have been discussed and re-emphasized.

A new concept of the inguina or hernia-producing region has been presented, which embodies the formerly recognized inguinal and femoral regions.

The inguina has been defined and portrayed as a pyramidal space consisting of a superior or extra-pelvic triangle and an inferior or intrapelvic

triangle, which are respectively divided into smaller anatomical triangular units.

Triangulation of the inguina does not conflict with existing basic anatomic concepts.

The erroneous but prevailing concept of defect as contrasted with weakness as it applies to plastic repair has been discussed.

The strategic advantages of the superior or inguinal approach over the inferior or femoral have been pointed out.

Attention has been directed to the value of an exclusively fascial stratification.

In the presence of a multiple locular sac, inadequacy of the inguinal ligament or diffuse parietal laxity, the iliopectineal (Cooper's) ligament hernioplasty is preferable.

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THYROIDITIS

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THERE ARE THREE MAIN AND DISTINCT CLINICAL TYPES of thyroiditis, (1) subacute (pseudo-tuberculous or giant cell) thyroiditis, (2) struma lymphomatosa (Hashimoto's thyroiditis), and (3) Riedel's struma (woody or ligneous thyroiditis). There are other types of thyroiditis that either have not been classified or represent incomplete or atypical forms of the above. In addition, thyroiditis may complicate bacterial or virus infections and may be the result of parasitic disease. It is not within the scope of this paper to discuss the rare and atypical types of thyroiditis but rather to define more clearly the recognized types and to discuss their treatment.

SUBACUTE THYROIDITIS (27 CASES)

Subacute thyroiditis is a self-limited disease of unknown etiology. It runs a variable course of weeks or months and eventually subsides without treatment and without significant interference with the function of the thyroid.

This type of thyroiditis has been variously named tuberculous, pseudotuberculous, or giant cell thyroiditis because of the histologic appearance of pseudotubercles with giant cells. Tubercle bacilli cannot be demonstrated in the lesions, and the etiology of the disease is unknown. Bacteria have not been demonstrated in the thyroid. The possibility that it represents a virus infection has not been excluded. The pseudotubercle or giant cell reaction represents a reaction of wandering cells to colloid, which they appear to be phagocytizing.

Many surgeons do not operate on patients with subacute thyroiditis and hence are not aware that this well-recognized clinical entity is, from the histologic standpoint, identical with pseudotuberculous or giant cell thyroiditis. In order to prove to my own satisfaction that the two diseases are the same I have analyzed 15 cases of subacute thyroiditis in which roentgen treatment was given and compared the history and physical findings with those of 12 cases in which operation was performed. To further confirm the fact that the clinical entity of subacute thyroiditis is indeed identical with the pathologic entity of giant cell or pseudotuberculous thyroiditis, biopsies of the thyroid were taken in two typical cases of subacute thyroiditis, and the patients were then treated with roentgen-ray. The biopsies showed typical giant cell or pseudotuberculous thyroiditis; the response to roentgen-ray was prompt, complete, and typical of that of subacute thyroiditis. In the table the two groups are compared, and it is clear that the cases are similar in most respects, the only difference being that the cases treated surgically were in general less acute and of longer duration. Many of these cases probably represent the subsiding phase of the disease.

Clinical Course. The onset of subacute thyroiditis is usually sudden and in about a fourth of the cases follows an acute upper respiratory infection. This history is difficult to evaluate because patients cannot invariably differentiate between a sore throat and a sore thyroid gland. It is six times more common in women than in men and tends to occur in the mid-forties.

Pain on swallowing and pain radiating up to the ear are characteristic of subacute thyroiditis. Usually the gland is exquisitely tender. A low grade elevation of the temperature is present (Fig. 1) and the sedimentation rate is elevated, often to high levels. There may be a marked systemic reaction.

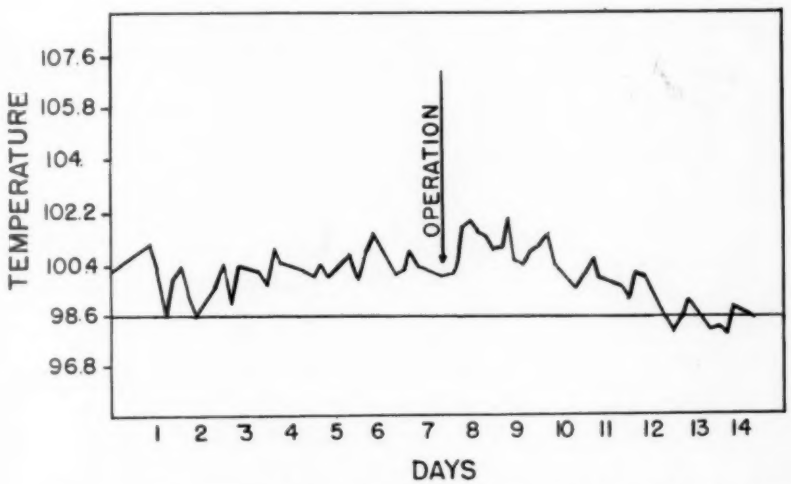

FIG. 1.—Temperature chart of patient with subacute thyroiditis. Before admission to hospital she had been observed for two months and had not improved. Only one lobe of thyroid was removed, but temperature and pulse rate fell promptly to normal.

The patient feels nervous, weak, and tired, and the pulse rate is elevated out of proportion to the temperature, sometimes as high as 160. Sweating and tremor are often prominent symptoms, so that the clinical picture may closely simulate hyperthyroidism. The basal metabolism, however, is not often elevated above the upper limits of normal and it is questionable whether true hyperthyroidism is present. The average basal metabolic rate is +10, but about one-third of the patients have basal metabolic rates over +15 per cent. The picture is that of a toxic reaction rather than of true hyperthyroidism. There is usually loss of weight, but since the duration of the disease is short the loss is slight. The eye signs of hyperthyroidism are not present.

Tenderness of the thyroid is almost always present, especially in the early stages when the gland is exquisitely sensitive to pressure. The entire gland is diffusely involved in most cases. Its consistency is abnormally firm or hard. Occasionally the process will start in one lobe and spread gradually to involve the entire gland ("creeping" type of thyroiditis).

The diagnosis of subacute thyroiditis usually is unmistakable and is suspected from the first. In only six of the 27 cases was the diagnosis missed by the first examiner.

The natural course of the disease appears to be toward spontaneous recovery without permanent derangement of the function of the thyroid. This course is shortened by thyroidectomy, roentgen-ray therapy, or it is said,[1] by treatment with thiouracil. This type of thyroiditis does not go on to either Hashimoto's or Riedel's disease, nor, in our experience, has suppuration occurred.

Pathology. Subacute thyroiditis is characterized by a diffuse involvement of the entire thyroid in a subacute inflammatory process. There is infiltration with leukocytes and numerous foreign body giant cells are present. The arrangement in formations somewhat resembling tubercles gives rise to the name pseudotuberculous thyroiditis. The foreign body reaction is probably a response to the colloid in the degenerating follicles, and histiocytes can be seen phagocytizing this material.

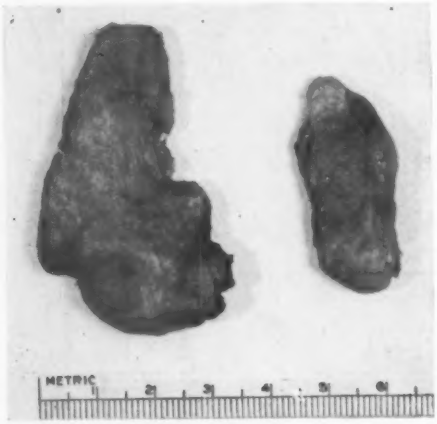

FIG. 2.—Gross appearance of subacute thyroiditis. The small adenoma at the upper pole is an unusual finding.

The thyroid rarely contains adenomas. The glands are not enlarged to more than two or three times their normal size, and the enlargement tends to be symmetrical. The cut surface of the gland is white and avascular. It is quite brittle and will not hold a hemostat. The capsule is only very lightly adherent to surrounding structures, and there is no tendency to infiltration and fixation, as in Riedel's struma. Tubercle bacilli have not been identified in this lesion (Fig. 2).

Treatment. Subacute thyroiditis responds promptly and completely to roentgen-ray therapy. Six hundred to 800 r usually suffice to effect a resolution in a few weeks. The pain and tenderness subside in a few days. The average time at which 15 patients treated with roentgen-ray were considered to be entirely well was 19 days after the start of treatment. By this time the thyroid is rarely either tender or palpably enlarged. In two of our cases, two or more courses of therapy over a period of three months were required before a complete cure was effected. Three patients considered themselves well in one week.

One patient developed a severe systemic reaction to an undulant fever skin test, and this was accompanied by an exacerbation of the pain and tenderness in the thyroid and was followed by complete resolution of the thyroiditis in a matter of a few days. Whether this was a specific or nonspecific

TABLE I.

	Reidel's Struma 11 Cases Treated by Partial Thyroidectomy	Struma Lymphomatosa 14 Cases Treated by Partial Thyroidectomy*	Subacute Thyroiditis 15 Cases (14 treated with roentgen-ray and 1 by undulant fever vaccine)	Giant Cell or Pseudotuberculous Thyroiditis; 12 Cases Treated by PartialThyroidectomy
Age	42 to 74; av. 51 (7 over 50)	30 to 72; av. 49 (8 over 50)	20 to 61. Av. 41	34 to 57. Av. 47
Sex	10 F, 1 M.	12 F. 2 M	14 F; 1 M	9 F; 3 M.
Duration	3 mo. to 7 yrs. Av. 29 mo. (8 were 1 yr. or more)	1 mo. to 8 yrs. Av. 20 mo. (7 were 1 yr. or more.)	Few days to 6 mos. Av. 6 wks.	3 wks. to 11 mos. Av. 11 wks.
Leading symptom	Pressure 6, goiter 2, nervousness 1, pain 2	Goiter 7, systemic symptoms 4, pressure 3 (pain at onset only 3)	15 pain in thyroid	Pain 11; no pain 1 (systemic symptoms)
Tender thyroid	1	0	15	10; not tender 2
Pain on swallowing	1	1 at onset only	12; no pain none; no statement 2	5; no pain 4, no statement 3
Pain radiating to ear	0	0	6; no ear pain 2; no statement 7	6; no statement 6
Temperature	Av 98.7°	Av. 98.6°	97.8° to 101°. Av. 99.4° (5 had temp. over 100°)	98.4° to 102.5°. Av. 99.9° (4 over 100°)
Pulse rate	80 to 120; Av. 88	64 to 128; av. 86	72 to 160. Av. 113 (6 over 120)	96 to 152. Av. 108 (2 had pulse over 120)
Loss of weight	Av. 9 lb.	Av. 6 lbs.	0 to 12 lbs. Av. 6 lbs.	0 to 30 lbs. Av. 13 lbs.
B.M.R.	−20 to +28; av. +2%	−24 to +18; av. −8%	−19 to +29. Av. +10	−13 to +41. Av. +10
Sedimentation rate	1 case 1.6	1 case 0.22	8 cases average 1.46 mm./min. (normal 0.4)	4 cases average 0.96
Diagnosis preop.	Thyroiditis 2; ca. or Riedel in 2; adenoma 1; ca. in 6	Thyroiditis 3, nodular goiter without hyperthyroidism 9, calcified adenoma 1, carcinoma or thyroiditis 1	15 correct diagnosis	9 recognized before op., 3 not recognized
Bilateral	5; unilateral 6 (2 of unilateral later involved the other lobe)	12, unilateral 2 (no biopsies of other lobe)	12; unilateral 3 (creeping type 2)	12; unilateral 0
Und. fever skin test	None tested	None tested	Neg. 4; strongly pos. 1, weakly pos. 1	2 cases, both neg.
Hyperthyroidism	None	None	None in 12; questionable in 3	None in 7, questionable in 5
History of upper resp. infect. at onset	1 questionable	None	3	4
Proved by exam. of tissue	11 (7 had associated adenomas)	14 (none had associated adenomas)	2 (biopsy only)	12 (2 had associated adenomas)
X-Ray therapy	3 cases; 2 with apparent arrest but no regression; 1 with no improvement after 2550 r.	1 case; no improvement; amount unknown	14 cases 300 to 1050 r. Av. 620 r. Well 1 wk. to 3 mos. Av. 19 days; all improved in 3 wks.	0
Surgery	Removal of part of each lobe 4; 3 pts. well, 1 hypothyroidism postop. Removal of part of 1 lobe 7; 3 pts. well; 2 developed recurrences in other lobe; 1 had persistent tumor in other lobe; 1 had persistence of spontaneous preop. tetany resulting from destruction of parathyroids by fibrosis	Thyroidectomy 10; 3 are well; 5 have hypothyroidism or some systemic disorder poorly controlled with thyroid; 1 has bilateral paralysis of vocal cords; 1 died postop. Lobectomy 4; 2 are well; 1 is improved but does not feel well; 1 has persistence of other lobe but feels well	Thyroidectomy 0 Lobectomy 0 Biopsy 2	Thyroidectomy 6; 5 well postop. Hypothyroidism 1 Lobectomy 6, 3 well post op.; 3 had symptoms due to persistence or recurrence in other lobe but eventually recovered.
Larynx	2 pts. developed spontaneous paralysis of vocal cord on affected side	No preop. paralysis	No laryngeal paralysis	No preop. Paralysis

*6 of the cases included in this series were previously reported by Dr. Allen Graham; ref. 2. 8 are unreported cases.

reaction to the undulant fever vaccine will never be known. Eight other patients had negative and one a weakly positive skin test for undulant fever.

Thyroidectomy is a satisfactory means of controlling subacute thyroiditis, but since the disease is essentially self-limited and since roentgen-ray effects such prompt and complete resolution, operation is not often indicated. Most of the patients reported here as having been subjected to operation were seen before we recognized the value of roentgen-ray.

Thyroidectomy was performed on six patients; five of these are well and one has developed hypothyroidism.

A single lobe was removed in six cases. Three of these patients developed a recurrence or suffered from persistence of symptoms due to involvement of the remaining lobe. All eventually recovered.

Since roentgen-ray has given entirely satisfactory results we have not used thiouracil in the treatment of subacute thyroiditis. If the foreign body reaction is indeed due to the presence of colloid, the beneficial action of thiouracil could be explained on the basis of its interference with the formation of this substance.

Case 1.—Subacute (pseudotuberculous or giant cell) thyroiditis.

The patient was a woman 32 years old. Two months before entry she had noted sudden onset of pain in the right side of the neck. This area was tender. There was palpitation, insomnia, nervousness, and an elevation of temperature to over 100°. She had lost six pounds in weight. Iodine had been given without improvement.

Examination showed a diffuse enlargement of the entire thyroid to one and one-half times the normal size. Both lateral lobes and the isthmus were stony hard and tender. The temperature was 99.6° and the pulse 108. There was a coarse tremor and the skin was dry. There were no eye signs of hyperthyroidism. The basal metabolic rate was +3 per cent.

A biopsy 2 mm. in diameter was taken from the isthmus of the thyroid and the pathologist reported chronic thyroiditis with marked granulomatous reaction to colloid (so-called pseudotuberculous thyroiditis). There was a fairly marked increase in connective tissue with the remaining follicles of small or medium size formed of flat or cuboidal epithelium, fairly well filled with colloid, and with some of the follicles partly filled by cells of the histiocyte or macrophage type. Colloid was markedly reduced or absent in the follicles containing these cells. Also present were fairly frequent accumulations of mononuclear cells and foreign body giant cells which in several instances enclosed small lakes of colloid. The stroma contained a slight to moderate infiltration of lymphocytes and some plasma cells and polymorphonuclear leukocytes (Fig. 3).

A total of 700 r of roentgen-ray was then given to the thyroid area in five treatments distributed over a period of nine days. At the end of this time the patient stated that she felt entirely well. There was no pain or tenderness of the thyroid, and the gland had returned to normal size, but the right lobe was still fairly firm. The consistency of the left lobe was normal.

Four weeks later the thyroid was soft and barely palpable, and the patient remained well.

Comment. The clinical features of this case and the response of the thyroid to roentgen-ray treatment are typical of those encountered in other cases of subacute thyroiditis. Biopsy of the thyroid showed changes characteristic of the so-called pseudotuberculous or giant cell thyroiditis. In a sec-

ond case subjected to biopsy the clinical course and response to roentgen-ray therapy were similar, and the pathologist reported a similar lesion in the thyroid. There can be little doubt that the clinical disease, subacute thyroiditis, is, from the pathologic standpoint, pseudotuberculous or giant cell thyroiditis. This disease responds promptly to roentgen-ray treatment and does not necessitate thyroidectomy.

STRUMA LYMPHOMATOSA (14 CASES)

Struma lymphomatosa is a progressive disease of the thyroid, possibly associated with systemic disorders,[2] in which there is extensive acidophilic degeneration of the epithelial elements of the thyroid and replacement by

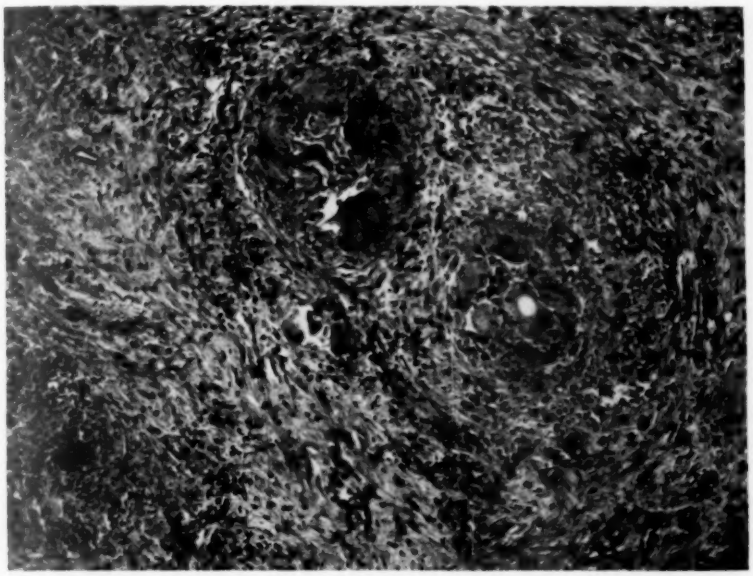

FIG. 3.—Subacute or giant cell thyroiditis. Biopsy obtained in case 1. Prompt and complete resolution following x-ray treatment (× 50).

lymphoid and fibrous tissue. Hypothyroidism or at least a peculiar type of hypometabolism that does not always respond specifically to desiccated thyroid is apt to develop. The etiology of the disease is unknown. It does not progress to Riedel's struma[3] nor is it the end result of subacute thyroiditis. An excellent description of this disease was given by Joll in the British Journal of Surgery in 1939.[4]

Clinical Course. Struma lymphomatosa occurs most commonly in the late forties or early fifties. Although it is rarely seen in men, two such cases are reported in this series.

True struma lymphomatosa is rare and is not to be confused with lymphoid infiltration of the thyroid or nonspecific types of lymphoid thyroiditis, from which it can be distinguished by the acidophilic degeneration of the epithelium. True Riedel's struma usually is somewhat more rare than struma

lymphomatosa. In my experience Riedel's struma and struma lymphomatosa are only about one-fifth as common as subacute thyroiditis. In the course of approximately 900 thyroidectomies I have records of two operated and 15 unoperated cases of subacute thyroiditis, three operated cases of struma lymphomatosa, five operated cases of Riedel's struma, and one of suppurative thyroiditis, as well as several cases that are unclassified.

The onset of struma lymphomatosa is insidious, and pain is noted only rarely at the onset. None of our patients had pain radiating to the ear, and only one had noticed pain on swallowing. The glands are not tender, there is no fever, and rarely is there any systemic reaction except that associated with hypothyroidism and a peculiar lack of feeling of well-being which does not always respond to treatment either by thyroidectomy or by administration of desiccated thyroid. It is interesting to note that in the only two patients who had gastric analyses there was no free acid. Graham observed anacidity or hypoacidity in six of his 14 cases. In one patient who died, postmortem examination showed generalized lymphoid hyperplasia. Mild anemia may be present.[2]

Half of the patients in this group complained merely of goiter. Four had systemic symptoms such as nervousness or loss of weight, and three complained of pressure symptoms from the enlarging gland. There was no consistent elevation of the temperature or pulse rate, and the basal metabolic rates averaged —8 per cent. In five of the 14 cases the basal metabolic rates were less than —10 per cent, —24 being the lowest.

The average duration of the goiter or of the symptoms prior to operation was 20 months. The sedimentation rate was normal in the only patient in which this was tested.

The entire gland usually is involved. In two cases it was stated that only one lobe was involved, but biopsies were not taken from the other lobe. The glands were described preoperatively as firm and "adenomatous." It is noteworthy that the gland does not appear to be so symmetrically involved in struma lymphomatosa as in subacute thyroiditis. Certain areas may enlarge more rapidly than others, giving a firm irregularity which in the majority of cases suggested the diagnosis of adenomatous goiter without hyperthyroidism. Once a calcified adenoma was suspected, once carcinoma or thyroiditis, and in only three cases was the diagnosis of chronic thyroiditis made before operation.

In three cases the tumor had enlarged to the point of causing tracheal compression. Most of the thyroids were four or five times normal size and when, as was occasionally the case, the growth encircled the trachea, symptoms of obstruction developed.

There does not appear to be any tendency to spontaneous remission or cure of this disease. One patient had had symptoms and an enlargement of the thyroid for eight years prior to operation. McClintock[5] has reported a case of struma lymphomatosa in which thyroidectomy was repeated two and a half years after the first operation and the histology of the gland was essentially unchanged.

Pathology. Graham[2] has said that there is no single clinical or pathologic feature of struma lymphomatosa that is characteristic or pathognomonic of this condition but that the entire clinical and pathologic picture, particularly the state of the thyroid gland as a whole, must be considered. Under these circumstances a fairly good case may be made out for either the Riedel or the Hashimoto type as a clinico-pathologic group, even if not an entity.

Struma lymphomatosa is characterized by acidophilic degeneration of the thyroid epithelium with replacement by lymphocytes and fibrous tissue. The lymphoid tissue often predominates and shows well developed germinal centers. There is no extension of the inflammatory process outside of the capsule and little or no tendency for the gland to become adherent to surrounding structures.

The thyroid is firm, friable, and not very vascular. Its cut surface is gray and lobulated and is sometimes mistaken for a hyperplastic goiter. Usually it is recognized as a thyroiditis at the operating table, but occasionally even pathologists fail to recognize it in the gross. The diffuse enlargement of the entire gland tends to form retrotracheal extensions which may render the gland difficult to deliver. In none of the 14 patients were there adenomas in the thyroid.

Treatment. It has been said that roentgen-ray treatment affords an effective means of controlling this type of thyroiditis.[6] Since we have rarely recognized the disease before operation we have not treated it with roentgen-ray. One patient was treated before she came to us by an unknown amount of irradiation without improvement.

Thyroidectomy was performed in 10 of the 14 patients. Three of the patients having thyroidectomy are well, five have hypothyroidism and require thyroid, one had a bilateral paralysis of the recurrent laryngeal nerves, and one died during operation with an unexplained convulsion. The high morbidity and mortality in this series suggests that conservative operations which do not attempt to remove all the gland may be preferable if the nature of the disease is apparent at the time of operation.

In four cases only a single lobe was removed. Two of these are well, one is improved, and one has a persistent enlargement of the other lobe but feels well. The record of lobectomy in this small group of cases appears to be better than that of thyroidectomy. Although roentgen-ray may be the treatment of choice, it will never be widely used unless the diagnosis can be made more often than it has been in the past. When struma lymphomatosa is first recognized during the operation it would seem best to perform a very conservative thyroidectomy, removing only enough of the isthmus and the lobes to relieve the pressure and leaving a moderate amount of thyroid tissue to help to prevent the development of hypothyroidism. This treatment is empiric and unsatisfactory, but unless we can establish the diagnosis before operation and until we know more of the etiology of the disease and of its response to roentgen-ray treatment it is the best available.

Case 2.—Struma lymphomatosa (Hashimoto). The patient was a woman 50 years of age who had been under treatment for three years for symptoms assumed to be due to the menopause. One month before entry her physician had noted an enlargement of the thyroid. She had noted tachycardia and palpitation, nervousness, and dyspnea on exertion. She had lost 6 pounds in weight.

Examination showed the temperature to be 98.3°, pulse 80, and blood pressure 160/90. There was a nontender, movable mass 6 cm. in diameter in the right lobe of the thyroid and slight enlargement of the left lobe.

The basal metabolic rate was 0. The red blood cells numbered 4,000,000 and the hemoglobin was 88 per cent.

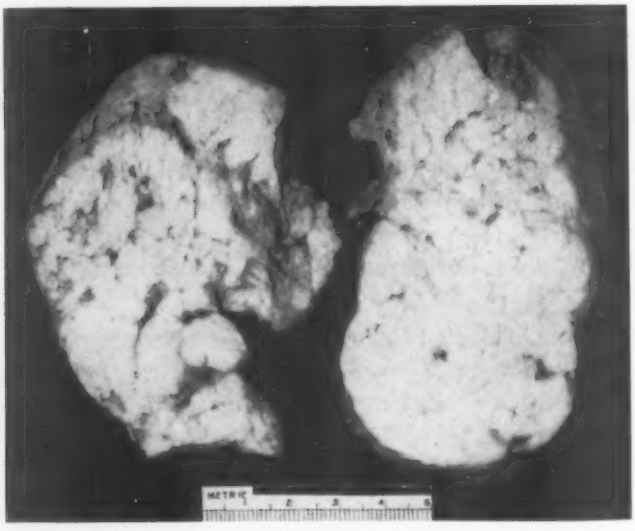

FIG. 4.—Gray color and lobulated appearance of struma lymphomatosa.

The preoperative diagnosis was nodular goiter without hyperthyroidism. At operation there was found a firm nodular goiter involving chiefly the right lobe. The capsule was only slightly adherent. There was a large retrotracheal and retrosternal extension of the right lobe. The gland was vascular, pale and firm. A diagnosis of struma lymphomatosa was made, and nearly all of the right lobe and part of the left lobe were removed. The left lobe was not as large as the right.

The specimen consisted of the greater part of the right and left lobes of the thyroid and weighed 90 Gm. (Fig. 4).

The thyroid epithelium was hypertrophic and acidophilic and there was great variation in the size and staining reaction of the nuclei. The colloid was diminished. The thyroid tissue was lobulated with slight increase of interlobular stroma and considerable lymphoid tissue distributed diffusely throughout and present also in numerous large hyperplastic lymphoid follicles. There were many plasma cells present (Fig. 5).

Convalescence was uneventful. Four months after operation the basal metabolic rate was —3 per cent, but the patient had the appearance of hypothyroidism and was given ½ gr. of desiccated thyroid daily. Fissures occurring at the corners of the mouth suggested a deficiency of vitamins.

Two years later the patient had no specific complaints but did not feel well. Thyroid feeding had not effected any improvement. Five years after operation the patient writes, "The effects of my operation are leaving me weak, but otherwise no trouble."

Comment. This case is typical in that (1) the symptoms before operation were poorly defined and vague, (2) the presence of a goiter was the chief complaint, (3) the preoperative diagnosis was adenomatous goiter without hyperthyroidism, (4) the true nature of the disease was first suspected at the time of operation, (5) after operation clinical evidence of hypometabolism and vitamin deficiency were apparent but did not respond to treatment, and (6) the patient still feels weak five years after operation.

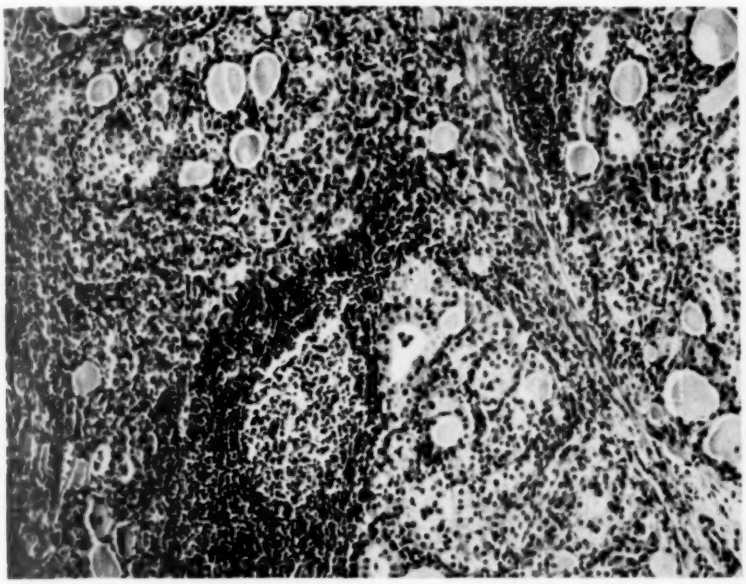

FIG. 5.—Case 2: Photomicrograph. Struma lymphomatosa (× 50).

RIEDEL'S STRUMA (11 CASES)

Riedel's thyroiditis is a chronic proliferating, fibrosing, inflammatory process involving usually one but sometimes both lobes of the thyroid and extending to involve the trachea and the muscles, fascia, nerves, and vessels in the vicinity of the thyroid. It produces a bulky tumor that may be indistinguishable preoperatively from an inoperable carcinoma. It is not the end result of either subacute thyroiditis or struma lymphomatosa but is a separate entity whose etiology is unknown. No specific organisms have been isolated from this lesion. In many instances the inflammatory reaction appears to center about a degenerating adenoma, and this may be a clue to its etiology.

Riedel's struma affects women more often than men and tends to occur beyond the age of 50.

Clinical course. The onset is insidious and usually painless. In no case did the pain radiate to the ears, and only one of the patients had pain on swallowing. Tenderness was present only once.

The tumor grows slowly, the average duration of the enlargement having been 29 months before operation.

Symptoms of pressure predominated in over half of the cases and often were severe with tracheal obstruction. Two patients had a symptomless goiter, and one patient complained only of nervousness. The temperature and pulse rate are not often elevated, there are few if any systemic symptoms, and little weight is lost. The basal metabolic rate usually is normal (average +2 per cent) although in one case, with almost total destruction of both lobes of the thyroid, hypothyroidism was present and the basal metabolic rate was —20 per cent.

The sedimentation rate was moderately elevated in the only case in which it was tested.

Characteristically, the tumor in the thyroid is localized to a part of the gland. In two of the unilateral cases the other lobe eventually became involved in the same process, once after a few months and once several years later.

The thyroid is stony hard and fixed to the surrounding tissues. Only two cases were correctly diagnosed before operation. In six cases carcinoma was suspected, in two cases the examiner could not decide between Riedel's struma and carcinoma, and in one case the preoperative diagnosis was adenoma. In all cases the true nature of the lesion was recognized at the time of operation.

In two cases a unilateral paralysis of the recurrent nerve developed spontaneously.

Pathology. In seven of the 11 cases adenomas or remnants of degenerating adenomas were present in the center of the proliferating fibrous tissue. Whether or not this finding is of etiologic significance I do not know, but in most of the specimens in which the major portion of the affected lobe was removed, degenerating adenomas were found. In several cases it was impossible to judge whether the major portion of the lobe had been removed, as the operation had been accomplished by piecemeal technic and the specimen consisted of innumerable chips of fibrous tissue.

The microscopic picture is of a chronic inflammatory reaction and replacement of thyroid by fibrous tissue. Bulky tumors five or six times as large as the original lobe are formed in this manner and these tumors infiltrate the capsule of the thyroid, the trachea, the muscles, the tissues of the carotid sheath, and the recurrent laryngeal nerves, in such a way as to render it impossible to find any natural plane of cleavage outside of the capsule of the thyroid. The disease is in reality a diffuse fibrosis of the neck with the thyroid at its center.

From the histologic standpoint there is nothing specific by which Riedel's thyroiditis can be recognized, but the gross appearance of the lesion is unmistakable.

The entire lobe of the thyroid is stony hard, adherent, and avascular. It can be cut in any direction without bleeding except from an occasional vessel which can be seen protruding from the fibrous tissue. The blood supply has been choked off by fibrosis. The gland is brittle and white, and cuts almost like cartilage. The difference between Riedel's and subacute thyroiditis is in the degree of destruction of the thyroid epithelium, the relative scarcity of

foreign body giant cells, in the extent of the extracapsular fibrosis, in the size of the gland, and in the fact that in Riedel's there is apt to be a degenerating adenoma at the center of the process. The fibrous tissue seems to be laid down in layers around this adenoma to form concentric rings, like an onion.

Treatment. An adequate trial of roentgen-ray therapy was made in three of these cases without significant results. In two cases in which one lobe had been removed and a recurrence later took place on the other side the roentgen-ray seemed to prevent further proliferations, although there was no change in the size of the tumor. In a third case, in spite of 2550 r. of roentgen-ray, pain and symptoms of compression continued, hypothyroidism developed, and the process extended to involve the parathyroids and produced tetany. We must assume, therefore, that roentgen-ray has little to offer in the treatment of this disease.

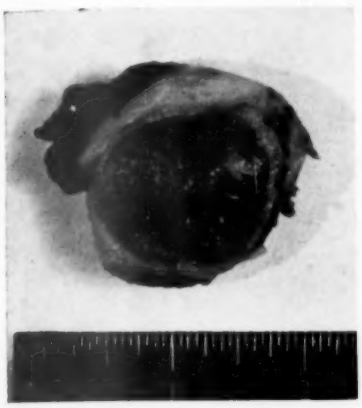

FIG. 6.—Case 3: Riedel's struma. The specimen represents only the portion of the lobe that was enucleated. Note adenoma encased in dense, concentrically laminated fibrous tissue.

Complete surgical removal of the involved portion of the thyroid may be rendered utterly impossible by the extent of the extracapsular fibrosis. Serious damage to the trachea, carotid sheath, or recurrent nerves may take place if the true nature of the lesion is not recognized and radial extirpation is attempted. In this disease one must often be content to do the best he can within the bounds of safety to relieve obstruction.

On the other hand, if one remembers the fact that in most cases, at the center of the fibrosed lobe, there is a degenerating adenoma and that around this adenoma the fibrous tissue is deposited in concentric laminations which afford natural cleavage planes, it is often possible, without jeopardizing the vital structures adherent to the capsule of the thyroid, to split the lobe open and enucleate this central core (Fig. 6). The results following this simple procedure have been excellent in the three cases in which I have found it practicable. Pressure symptoms have been relieved, the bulk of the tumor has been strikingly diminished, and the progress of the inflammatory and productive process appears to be arrested. It is well to remember that the most severe obstruction to respiration usually is associated with retrotracheal adenomas that compress the trachea from behind and that this can be demonstrated before operation by a lateral roentgenogram of the trachea. If the surgeon is not aware of the retrotracheal tumor he is apt to overlook it in a thyroid which cannot be mobilized and rotated from its bed.

Six of the 11 patients who had portions of the thyroid removed for Riedel's struma are well. In seven patients only one lobe was removed, and in two of these the process recurred on the other side. One patient had bilat-

eral involvement and has a symptomless persistence of the disease after removal of part of one lobe. One patient has hypothyroidism, and one chronic tetany which developed spontaneously before operation, probably as a result of destruction of the parathyroids. The thyroid is so extensively destroyed that this patient also has hypothyroidism.

Case 3.—The patient was a woman 46 years old. She had noticed an enlargement of the left lobe of the thyroid two years before entry, and a few months later she became

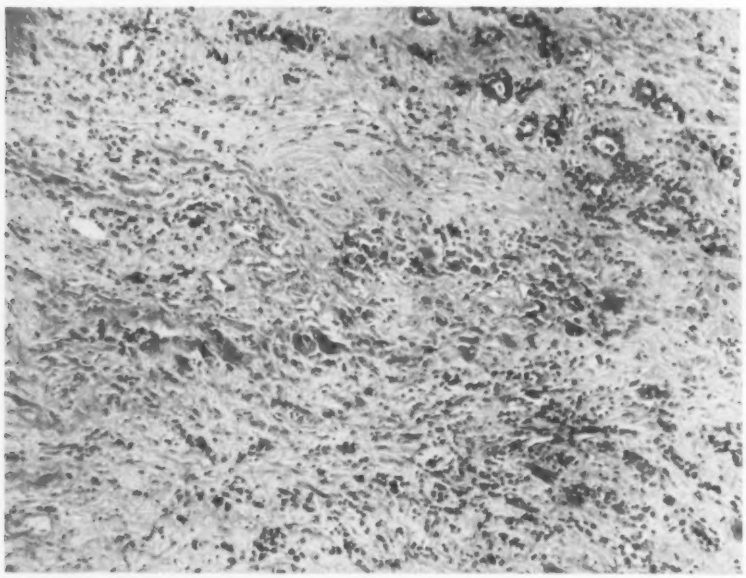

FIG. 7.—Case 3: Photomicrograph. Riedel's struma (× 50).

hoarse. Three months before entry an attempt was made to do a thyroidectomy, but the surgeon stated that the tumor was an inoperable carcinoma. A biopsy was interpreted as either Riedel's struma or carcinoma.

Symptoms of pressure continued, but there was no loss of weight and no symptoms of systemic disease.

Examination showed a stony hard, fixed tumor involving the left lobe of the thyroid and the cervical musculature. The left vocal cord was paralyzed. The basal metabolic rate was +2 per cent.

A diagnosis of Riedel's struma or inoperable carcinoma was made. At operation the entire left side of the neck was involved in a dense proliferation of fibrous tissue which had invaded the trachea and the prethyroid muscles. The lobe was exposed and the anterior half removed. It was white, brittle, and avascular. The cleavage plane surrounding the central adenoma was located, and a degenerating adenoma was shelled out in a capsule of fibrous tissue (Fig. 6). The right lobe was normal.

The pathologist reported chronic inflammation and complete replacement of thyroid tissue with fibrous tissue (Fig. 7). There was a degenerating colloid adenoma in the center of the fibrous mass.

Convalescence was uneventful and the symptoms of pressure were completely relieved.

Comment. This case is typical of Riedel's struma in that (1) the initial diagnosis was carcinoma of the thyroid; (2) there was no systemic reaction or alteration of thyroid function, (3) there was extensive fibrosis of the thyroid and of the perithyroid structures, (4) there was a degenerating adenoma in the center of the fibrous mass, (5) symptoms were relieved by enucleation of the adenoma from the surrounding fibrous tissue.

OTHER TYPES OF THYROIDITIS

During the same period of time in which these cases of specific thyroiditis were seen, there have been at least 10 cases of well-defined thyroiditis which do not fit into any definite category. There have also been three cases of suppurative thyroiditis occurring in degenerating adenomas. I have seen no proved syphilis or tuberculosis of the thyroid.

CONCLUSIONS

1. The clinical entity described as subacute thyroiditis has been proved by biopsy to be giant cell or pseudotuberculous thyroiditis.

2. Subacute thyroiditis, struma lymphomatosa, and Riedel's struma are separate clinical entities and probably are etiologically unrelated to one another, or at least do not represent various stages of the same disease.

3. The fact that subacute thyroiditis is almost always associated with pain and tenderness and that these symptoms only rarely occur in the other types argues against the possibility that subacute thyroiditis represents an early stage of the more chronic processes. The tendency to spontaneous recovery in subacute thyroiditis and its prompt and dramatic response to roentgen-ray treatment also argue against this possibility.

4. The fact that Riedel's struma is more often unilateral and struma lymphomatosa usually involves the entire thyroid,[3] and the frequent presence of adenomas in Riedel's while they are rarely if ever recognized in struma lymphomatosa is further argument against progress of one lesion to the other. It is inconceivable that the fibrosis of Riedel's struma could regress and become a struma lymphomatosa.

5. Although the etiology of these diseases is unknown, it is possible that subacute thyroiditis is the result of a virus infection and that persistence of symptoms and evidence of inflammation in the thyroid is due to a foreign body reaction to colloid.

6. Struma lymphomatosa appears to be a systemic disease, possibly of the deficiency type, and further study of its relationship to achlorhydria, anemia, generalized lymphoid hyperplasia, and possibly to vitamin or other deficiencies is indicated. Many of these patients are not well before or after operation, and the hypometabolism and associated symptoms may not be specifically corrected by feeding desiccated thyroid.

7. Riedel's thyroiditis appears to be a proliferative fibrosis usually centering about a degenerating adenoma. Although the role of this adenoma cannot

be proved, it is possible that some change in the adenoma sets off a fibrous tissue reaction resembling that seen in a keloid. Removal of the core containing the adenoma appears to promote subsidence of this reaction.

8. Roentgen-ray is the treatment of choice for subacute thyroiditis and thyroidectomy is rarely if ever indicated. Thiouracil may be of value.

9. In the rare cases of struma lymphomatosa in which the diagnosis is made before operation roentgen-ray should be given a trial. If struma lymphomatosa is recognized at the time of operation a very conservative resection of both lobes of the thyroid is recommended. The morbidity of radical resection is high and postoperative hypometabolism the rule when most of the gland is removed.

10. In Riedel's struma roentgen-ray is of little or no value, and surgery is apt to be difficult. It is unwise, unnecessary, and often dangerous to attempt to remove the entire lobe. If the onion-like concentric laminations in the fibrous tissue surrounding the central degenerating adenoma can be found, these avascular planes can be followed by blunt dissection and the core of the lobe shelled out without disturbing its capsule. Following this procedure the symptoms are relieved and there is no further proliferation of fibrous tissue.

NOTE: Since this article was prepared, 2 patients with struma lymphomatosa proved by biopsy have been treated by roentgen-ray. Fifteen hundred roentgen units was given. In 1 case the thyroid enlargement resolved promptly and was barely palpable at the end of three weeks. In the other the response was slower, but at the end of 6 months the thyroid was not palpable. In neither case was there improvement of the systemic symptoms.

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COARCTATION AND ANEURYSM OF THE AORTA

Report of a Case Treated by Excision and End-to-End Suture of Aorta

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IN 1944 ALEXANDER AND BYRON[1] reported a case in which an aneurysm of the descending aorta was treated by excision, with proximal and distal ligation of the aorta. This was the first recorded instance of resection of an aortic aneurysm. Their patient was a 19-year-old boy in whom a diagnosis of coarctation of the aorta was made, based upon the presence of hypertension in the upper extremities, weak arterial pulsations in the lower extremities, and well-developed collateral circulation evident during the operative dissection, and demonstrable by typical notching of the ribs on roentgenographic examination. Though the excised specimen did not include the area of stenosis the diagnosis of coarctation seemed well-established.

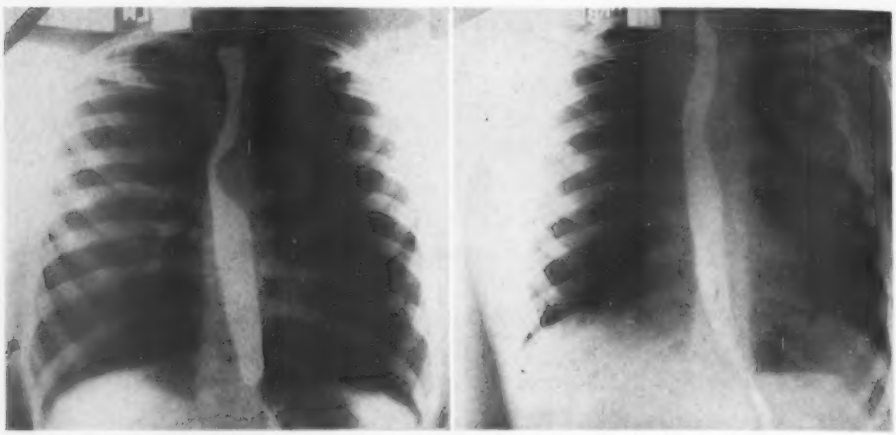
Studies in dogs in which an induced coarctation was treated by end-to-end anastomosis of the subclavian artery to the aorta below were reported by Blalock and Park in 1944.[2] In 1945 Gross and Hufnagel[3] demonstrated experimentally the feasibility of excising a segment of aorta and repairing the defect by end-to-end suture. Shortly afterwards clinical cases of excision of the coarcted aorta with end-to-end repair were reported by Crafoord and Nylin[4] and by Gross.[5] Though only a few instances of surgical correction of coarctation have been reported thus far, the procedure is being utilized more frequently as time passes and with gratifying results.

The purpose of this report is to record an unusual case of coarctation of the aorta associated with an aneurysm distal to the stenosis treated successfully by excision and end-to-end repair of the aorta. So far as I am aware, there has been reported no similar instance of successful extirpation of an aortic aneurysm by excision and resuture of the divided aorta.

CASE REPORT

The patient was an 8½-year-old boy in whom a cardiac murmur had been detected at the age of 2 years. His mental and physical development had been normal but his general activity had been restricted because of the presence of the murmur. He had enjoyed good health up until the past year during which he had had several attacks of otitis media. On January 9, 1947, he developed an earache and a fever of 103.5°. Three days later he was admitted to the Bridgeport Hospital with a tentative diagnosis of rheumatic fever. On the day of admission, as well as on the 4th and 9th days after entry into the hospital, blood cultures were positive for pneumococcus Type VII. Salicylate therapy had been instituted upon admission and on the 4th day treatment with penicillin, 40,000 units every two hours, was begun. Because of the bacteremia, the cardiac murmur, and the continued septic fever, it was thought that he had bacterial endocarditis. On January 23 he was transferred to the Pediatric Service of the Grace-New Haven Community Hospital.

The patient was a well-developed, rather slender boy, with a temperature of 101° F. and a pulse rate of 104. There was a loud blowing basal systolic murmur heard best to the left of the sternum in the first, second and third interspaces and also heard well posteriorly, especially to the left of the vertebral column. Some observers thought there might be a faint diastolic murmur though others could not hear it. Both tympanic membranes were dull but there was no aural discharge. General examination was not remarkable except for the murmur, the fever, and a brachial blood pressure of 140/70. The initial impression was that the patient had congenital heart disease and bacterial endocarditis. He was treated with penicillin 100,000 units every 2 hours and sulfadiazine 0.5 Gm. every 4 hours. Blood cultures were negative and treatment was discontinued after 6 days. Roentgenograms of the chest showed notching of the ribs and questionable left ventricular enlargement. It was then noted that the pulses were weak or absent in the



A B

FIG. 1.—Roentgenograms of the chest during barium swallow. The heart size is considered normal; it is perhaps slightly more globular in shape than is usually seen. On these and other films there is questionable evidence of slight left ventricular enlargement. Notching of the ribs is seen. A. Preoperative roentgenogram. There is marked indentation upon the esophagus by the aneurysmal mass. B. Roentgenogram taken on 12th postoperative day. The circular indentation of the esophagus seen in the preoperative film is not evident.

lower extremities. Some observers thought they could palpate a feeble femoral and popliteal pulsation; I could not feel them. The dorsal pedal and posterior tibial pulses were absent. It was apparent that the patient had coarctation of the aorta. Though there was a possibility that the bacteremia had resulted from aural infection, it was felt that it might well have originated from bacterial aortitis. The patient was discharged on February 3.

On February 20 he was re-admitted. He had remained well. The systolic murmur was again noted. The right brachial blood pressure was 150/104, the left 154/104. The femoral and popliteal pulses were absent or very feeble. The dorsal pedal and posterior tibial pulses were absent. Venous pressure in the left arm at heart level was 105 millimeters of water. Macasol circulation time from arm to tongue was 12 seconds, to the body 17 seconds, and to the right foot 25 seconds. The urine was normal, the erythrocyte count 4.6 million, the hemoglobin 12.5 Gm., the leucocyte count 7,400. Hematocrit was 39 and sedimentation rate 14 millimeters in 30 minutes. Plasma proteins were 7.3 Gm. per cent and chlorides 103.8 milli-equivalents. The oral temperature ranged from 98.6° to 100.6° F. Electrocardiograms revealed evidence of a slight left axis deviation. Roentgenograms showed a left aortic arch. Though the diagnosis was not made before oper-

ation, in retrospect the roentgenograms with barium swallow (Fig. 1A) revealed evidence of a mass in the region of the first part of the descending aorta suggestive of an aneurysm.

On February 24, operation was carried out under intratracheal ether-oxygen anesthesia. A curved incision was made posterior to and below the left scapula. Numerous large collateral vessels were encountered in the subcutaneous and muscle layers. The fifth rib was resected subperiosteally from the transverse process out to the axillary line and a short posterior segment of both the 3rd and 4th ribs was excised. The aortic arch appeared to be somewhat smaller than normal. There was moderate dilatation of the left common carotid and marked dilatation of the left subclavian artery. The latter was about 1.3 cm. in diameter at its base, about the same size as the arch of the aorta. Just a few millimeters beyond the origin of the subclavian artery there was a marked narrowing of the aorta (Fig. 2). Beyond this point the thoracic aorta was a little more than

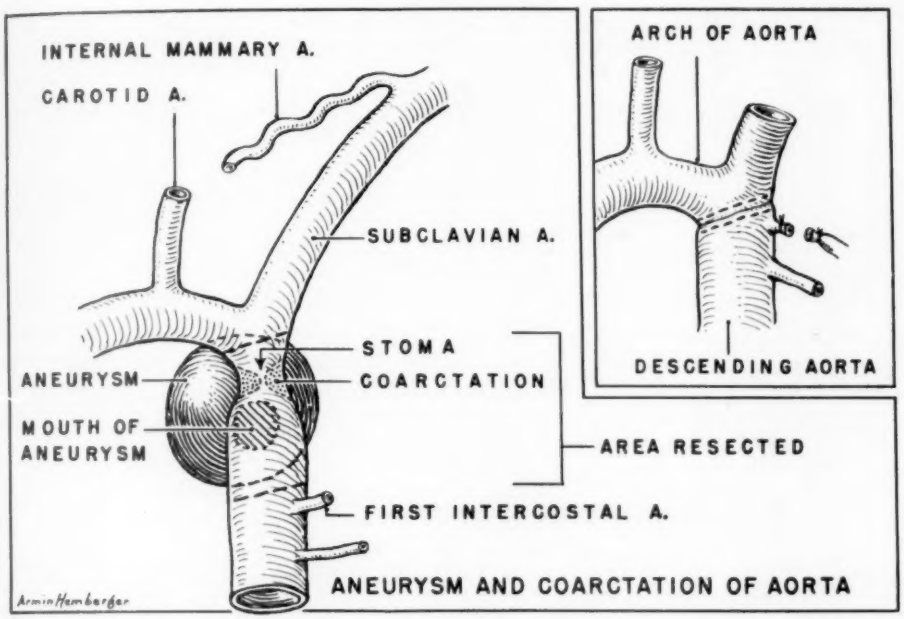


FIG. 2.—Diagrammatic sketch of the condition found at operation. The condition following excision of the aneurysm and coarcted portion of the aorta is shown in the insert.

2 cm. in diameter. Here a marked systolic thrill was felt but there was no visible or palpable pulsation. The arch of the aorta and the vessels arising from it pulsated vigorously. The first left intercostal artery was markedly dilated and tortuous, the second slightly less enlarged and the third only a little larger than normal. The internal mammary artery was tortuous and much enlarged, being about 6 mm. in diameter.

The first and second left intercostal arteries were dissected free in their proximal portions and, since it appeared essential for proper mobilization of the aorta distal to the coarctation, the first was divided between ligatures. Next the ligamentum arteriosum was isolated and divided between ligatures; it appeared to be obliterated. It was apparent that the coarctation existed in the area of the aorta connected with the ligamentum arteriosum. Dissection here and along the mesial and posterior aspects of the aorta was very difficult and tedious because of the presence of an aneurysmal mass almost twice the diameter of the aorta itself. It appeared to rise just below the coarctation and protruded against the esophagus mesially. The aneurysm was carefully freed except for the most

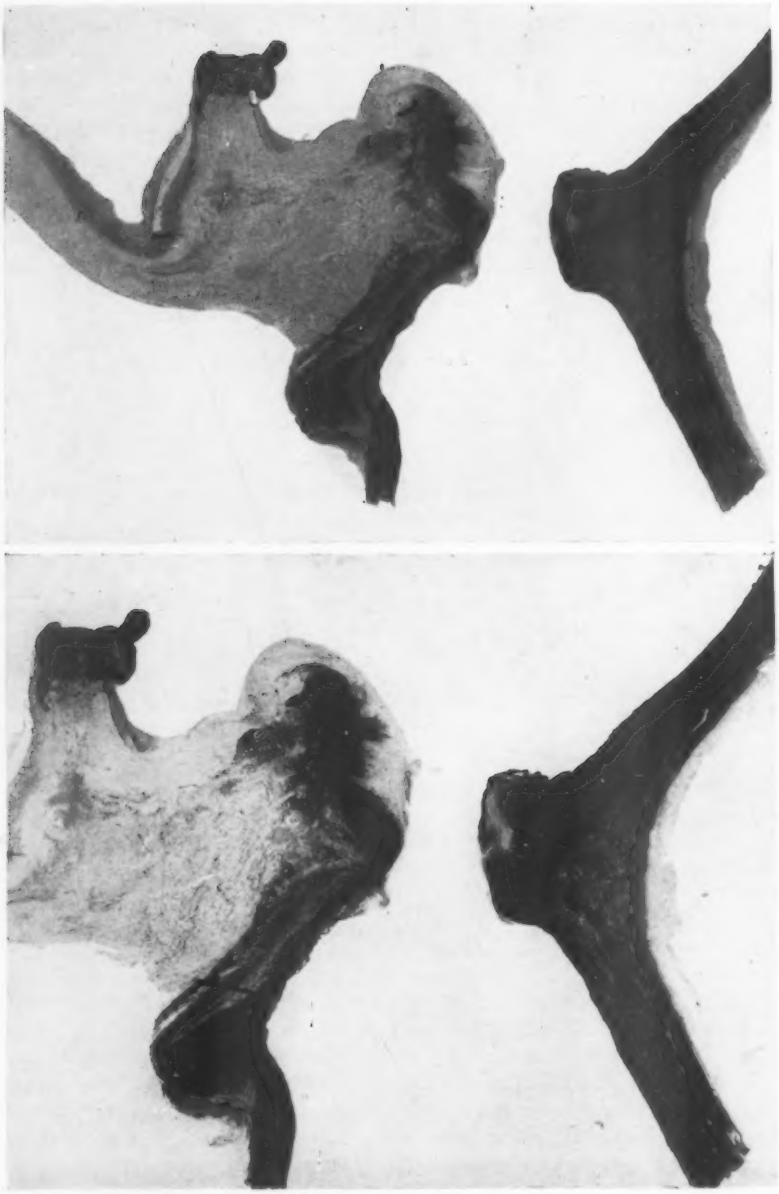
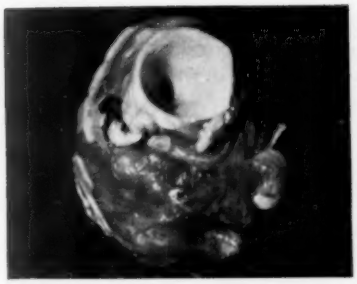


FIG. 3—Low power photomicrographs of section through aorta and aneurysm. Werhof elastic stain. The proximal aorta is seen below. Distal to the coarctation the wall of the aorta opposite the aneurysm appears normal but the aneurysmal wall has very little elastic tissue except for the knotted fibers on the ridge seen to the left of the edge of the mouth of the sac.

A



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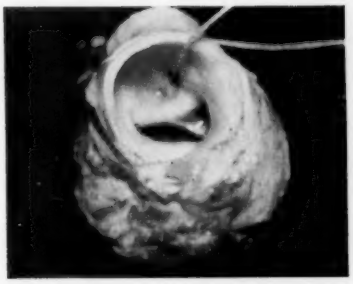

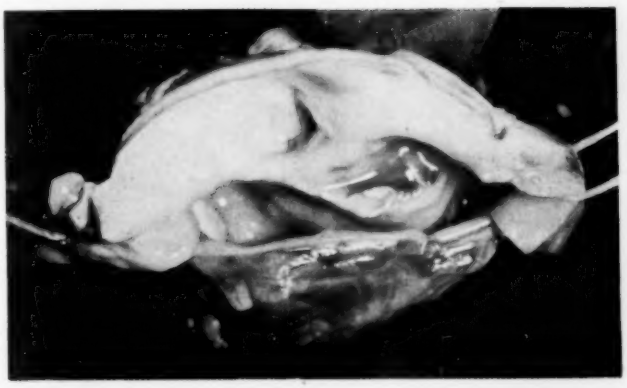

C

PLATE 1. Kodachrome photographs of the excised specimen. (A) The proximal end of the excised segment of aorta with the small aperture through the stenotic area and the aneurysmal sac below. (B) The distal end of the excised aorta. The opening from the aorta into the aneurysm is seen. The black silk ligature is on the stump of the ligamentum arteriosum. (C) The aorta and sac opened. The small beady protrusions within sac wall suggest vegetations.

mesial portion which I felt could not be separated safely from the esophagus and adjacent tissues until the aorta was clamped. The first and second right intercostal arteries had to be ligated and divided in order to mobilize the aorta properly. The subclavian artery and the distal end of the arch were mobilized. Special aorta clamps were now applied above and below the coarctation and the aneurysmal mass. In order to prevent occlusion of the subclavian artery the clamp was placed tangentially across its origin and even so a rather short segment remained between the clamp and the stenotic portion of the aorta. With the clamps in place the sac was now completely freed without rupture. About 2.5 to 3 cm. of the aorta were excised, including the area of coarctation and the aneurysm. The two ends of the aorta were approximated with a continuous everting mattress suture of number 5 Deknatel, interrupted in several places. Since the proximal aorta was smaller than the distal, it had been sectioned tangentially in order to make its divided end more nearly the size of that of the distal segment. The clamps were released slowly with the patient in Trendelenburg position and during rapid intravenous administration of blood. No significant fall in blood pressure occurred. Two small leaks on the posterior wall at the line of repair were stopped by additional sutures; the area was now dry. The distal aorta pulsated vigorously. The mediastinal pleura was approximated with interrupted silk sutures and, following, inflation of the lungs, the wound was closed in layers with silk sutures.

The stoma through the stenotic portion of the aorta had a diameter of only 1.5 mm. (Plate I). Just distal to the coarctation on the posteromesial aspect was an opening about 1 cm. in diameter into an aneurysmal sac of about twice the diameter of the aorta itself. Within the sac were present small pearly protrusions which looked very much like vegetations. The aorta showed no significant microscopic changes except in the area of stenosis and in the region of the aneurysm (Fig. 3). The fibro-elastic media appeared well preserved. At the site of the coarctation the subendothelial fibrous connective tissue was reduplicated, there was a thick layer of dense scar tissue, and the fibro-elastic media was largely replaced by fibrous connective tissue which extended in strands into the proximal and distal aorta. Here the laminations were distorted with separation of the fibrils by an increased amount of fibrous connective tissue. Opposite the mouth of the aneurysm the media of the aorta was normal in appearance. At the opening of the aneurysm the elastic tissue ended abruptly and the elastic fibers were coiled into knotted and entangled fragments. The sac itself was composed of dense collagen fibers, forming a connective tissue wall which was infiltrated with many plasma and mononuclear cells especially about the blood vessels. There were rare scattered thin atrophic elastic fibers here and there in the wall of the aneurysm; otherwise no elastic tissue was present except in the region of a fold upon which the vegetation-like lesions were centered. Here there was a dense mass of knotted elastic tissue fibers external to which an area of necrosis with numerous polymorphonuclear leucocytes was seen in the wall of the sac, and internal to which was dense fibrous connective tissue. The entire sac was lined with a thin layer of blood platelets and fibrin. Bacterial stains demonstrated no organisms. The blood vessels in the wall of the sac showed occlusion of the lumen by proliferation of endothelial cells, connective tissue replacement of the musculature, and fragmentation of elastic tissue.

The patient had an uneventful convalescence. Vigorous femoral, popliteal, dorsal pedal and posterior tibial pulses were present immediately after operation and popliteal blood pressure was 124/90. The following day blood pressure in the thigh by auscultation of the popliteal vessels was 140/100; blood pressure in the arm 112/90. Twenty-thousand units of penicillin were administered every 3 hours for 10 days. Except for an elevation to 101° on two occasions, the temperature was never over 100° and soon became normal. The patient was kept in an oxygen tent during the first day and night. He was allowed up in a chair on the 4th day and began to walk on the 8th day. His urine and blood count were normal. On occasions a faint precordial systolic murmur was

present, although it was sometimes inaudible. Roentgenograms with barium swallow showed an absence of the mass previously demonstrated (Fig. 1B). The patient was discharged on February 10 and was allowed to be normally active. On discharge blood pressure in the right arm was 118/86, left arm 112/90; right thigh 134/90, left thigh 130/94. Oscillometric studies revealed normal values, 5.5 to 6 in the thighs, 3 to 4 in the legs. The patient remained well.

He was re-examined on April 26. He had gained 10 pounds during the past 2 months. Heart rate was 75. There was a short systolic murmur heard to the left of the sternum centered about the 4th interspace. Blood pressure was 106/70 in the right arm, 102/70 in the left arm; 108/70 in the right thigh, 104/70 in the left thigh. Femoral, popliteal and posterior tibial pulses were full and equal. Oscillometric studies again showed normal values, 5.5 and 6 in the thighs, 4.5 and 5 in the legs, and 1.2 and 1.4 in the ankles.

DISCUSSION

Several interesting problems are presented in connection with the case reported. The first deals with the coexistence of coarctation and aneurysm of the aorta and of coarctation and bacterial aortitis. As Abbott[6] pointed out in her review of 200 cases verified at postmortem examination, dilatation of the ascending aorta is very common. This was the finding in 101 cases, while in 7 the aorta was normal in size, and in 21 hypoplastic. No comment concerning this matter was found in the records of 71 cases. She observed that the state of dilatation varied greatly from case to case and that in some a diffuse or saccular aneurysm resulted, or a dissecting aneurysm which, in turn, sometimes ruptured into adjacent structures. She gave no data concerning the incidence of expansion of the descending aorta but remarked that "localized dilatation of the descending thoracic aorta *immediately below* the stenosis is common." The enlargement of the descending aorta is often a bulbous dilatation with a normal sized aorta below the 4th or 5th intercostal. In her survey, 7 examples of saccular or spindle-shaped aneurysm distal to the site of coarctation were mentioned; in three of them rupture with fatal hemorrhage ensued. Nine instances of mycotic aneurysm beyond the coarctation were mentioned in three of which rupture occurred.

The recent review of 104 additional autopsied cases of coarctation by Reifenstein, Levine and Gross[7] records no specific data concerning the incidence of aneurysms. They stated, however, that dilatation of the aorta was commonly observed in the ascending and, less commonly, in the descending portion. It is of interest that the incidence of rupture of the aorta as a cause of death in their cases was nearly the same as in Abbott's series (23.1 per cent as compared with 20 per cent) and that bacterial endocarditis or aortitis caused more deaths in their series than in Abbott's (22.1 per cent as compared with 16 per cent).

Rupture of the aorta apparently bears a direct relationship to aortic dilatation or disease. Abbott noted death from rupture of the ascending aorta in 33 cases and at or near the site of coarctation in five cases. Referring to the 33 cases of rupture of the ascending aorta she pointed out that the aorta was markedly dilated in 29 and frequently was the site of an aneurysm; in the

great majority a dissecting aneurysm had formed with subsequent rupture. Reifenstein *et al* observed rupture of the ascending aorta in 19 cases and of the descending aorta in five. Among 14 cases of mycotic endarteritis in Abbott's series the infection was at the seat of coarctation in 13, while one was a mycotic aneurysm of the ascending aorta. Nine of the cases of endarteritis in the region of the coarctation had mycotic aneurysms. Similarly Reifenstein and his associates observed six deaths from bacterial aortitis, which in two cases affected the ascending aorta and in four the aorta distal to the coarctation. Their survey included one case of spontaneous recovery from bacterial aortitis followed by progressive aneurysmal dilatation and calcification of the affected portion of the aorta, possibly mycotic in origin. They also mentioned another case in which an alpha streptococcal bacteremia was presumably cured by penicillin therapy with subsequent aortic dilatation distal to the coarctation. It is well-known that limited rupture of a vessel with production of a non-fatal dissecting aneurysm may result eventually in a lesion indistinguishable from any other saccular aneurysm. Their report included one case of a dissecting aneurysm distal to a moderate coarctation which showed at postmortem examination 19 months later an "endothelialized" lumen.

Several factors may contribute to the dilatation of the aorta and to the formation of an aneurysm or rupture. With regard to the ascending aorta the question of hypertension and the relative increase in intra-aortic tension due to stenosis at the seat of coarctation must be considered. Gross and microscopic evidence of alterations in the wall of the aorta have been observed. Though Reifenstein and his co-workers deplored the infrequency with which case reports included careful microscopic studies of the aorta at the site of rupture and below and above the coarctation, they pointed out that decrease in the thickness of the media was generally noted, with necrosis, hyaline degeneration, fibrosis, decrease in and fragmentation of the elastic tissue, and cystic change. They also pointed out that atheromatosis was common, that the vasa vasorum were occasionally narrowed, and that the outstanding alteration was destruction of elastic tissue. The relative roles of hypertension and of congenital changes of the aortic wall in the production of these lesions is not well-established. Abbott spoke of several factors which may influence the development of an aneurysm distal to the coarctation. She stated that the bulbous dilatation just beyond the coarctation was a "direct result of the return of the collateral circulation through the aortic intercostals." She also pointed out that "even where this localized increase in diameter is not marked, the aorta just below the constriction at the entrance of the collateral blood often shows a patchy atheroma, which may act as a predisposing factor for the formation of a dissecting aneurysm with spontaneous rupture in this situation or for the development of a mycotic aneurysm." In addition she stated that in other cases traction by the ligamentum arteriosum produces a kinking or outpouching of the right lateral wall of the aorta, and that this area may rupture externally.

In my case the aneurysm was not recognized before operation. The patient had no complaints attributable to such a lesion and ordinary chest roentgenograms revealed no mass suggesting an aneurysm. Roentgenograms taken during barium swallow, however, showed a large esophageal indentation which was hardly compatible with the usual impression made by the aortic knob. The laminograms demonstrated a mass near the tracheal bifurcation which was absent after operation and which was much the same in size, shape and position as the aneurysmal mass. It was evident in retrospect that these observations should have established the diagnosis before operation.* Examination of the excised specimen gave no specific clue as to the etiology of the lesion. It was similar in gross and microscopic appearance to the usual saccular aneurysm produced by disease of the arterial wall or by trauma and to the ordinary mycotic aneurysm after subsidence of infection. There was no bulbous dilatation of the first part of the descending aorta, so that general thinning of the wall from such a process is not a likely explanation for the production of the lesion. It was situated on the right lateral wall of the aorta but since the ligamentum arteriosum was attached several millimeters proximally at the site of the stenosis, it appears unlikely that traction by this structure was a factor. The mouth of the sac was in an area where atheromatous plaques are common and it is entirely possible that the giving way of such a lesion may have produced the aneurysm. The patient had a pneumococcal septicemia and the sac was lined with an irregular organized thrombus which grossly resembled a vegetative process. Though no organisms were demonstrable, the microscopic picture was not incompatible with an infected vegetation which had been rendered sterile by penicillin. Whether the bacteremia resulted from such a process or from the mild otitis media cannot be established. It seems unlikely that a bacterial aortitis at the time of the bacteremia could have produced a mycotic aneurysm which would have such a well-organized gross and microscopic appearance 7 weeks later. It is entirely possible, of course, that the lesion may have developed asymptomically as a mycotic aneurysm some time in the past.

The second problem raised by the case reported concerns the treatment of aneurysms of the aorta in general and in particular those of the descending thoracic aorta. Bigger,[9] Elkin,[10] Alexander and Byron, and de Takats and Reynolds[11] have recently reviewed the literature concerning the operative treatment of aneurysms of the aorta and have pointed out how few are the cases of successful results from ligation. As far as I can ascertain only in the case of Alexander and Byron and in the case which I have described has an aortic aneurysm been excised, and only in the latter case has the aorta been repaired by end-to-end suture. Attention should be called to the fact that

* Clark and Koenig[8] have recently reported a case of saccular aneurysm distal to the area of coarctation proved at autopsy which was recognizable on roentgenographic examination because of calcification of the sac wall. They presented a second probable case of distal aneurysm in which a shadow was visible on ordinary films of the chest.

recently two cases of arteriovenous fistula of the abdominal aorta have been treated successfully by closure of the arterial defect with maintenance of the continuity of the aorta.[12, 13] In general it has been necessary to treat aortic aneurysms by some method such as internal wiring and coagulation. Some excellent results have been obtained with this method[14, 15] and I have been reasonably well satisfied with most of the cases which I have treated in this manner. Unless this procedure can be combined with complete aortic occlusion, however, it must be looked upon as a palliative rather than a curative measure.

In speculating upon the feasibility of excision of aortic aneurysms the problem of collateral circulation immediately comes to mind, and it is of interest that the only two cases thus far so treated have been instances of aneurysm in conjunction with coarctation. It would, of course, be possible to excise an aneurysm and ligate the severed aorta only if well-developed collateral circulation were present. Even then one would be concerned with the production of hypertension unless the ligation were carried out in the distal aorta. Indeed, without adequate collateral circulation one might be fearful of occluding the aorta temporarily in cases in which it appeared likely that restoration of continuity of the aorta might be accomplished, because of the hazard of ischemic damage to the spinal cord, a complication often noted after temporary aortal occlusion in dogs. Only further experience will reveal whether the aorta can be safely occluded temporarily in the absence of abundant collateral circulation and for how long. To be sure, Crafoord has often clamped the aorta in cases of patent ductus arteriosus during closure of the aortic end of the ductus without harmful effect. He informs me that up to June 1947 he has thus completely occluded the aorta for intervals of from 12 to 28 minutes in 31 patients without difficulty; only in one instance in which the occlusion was maintained for 48 minutes did symptoms of spinal cord injury follow.

The problem of bringing about development of adequate collateral circulation around a segment of aorta has been discouraging due to the tendency of external bands and ligatures to cut through the wall of the vessel in time, with resultant fatal hemorrhage. Blakemore has, however, recently made an important contribution in demonstrating that the diameter of the aorta can be safely narrowed by progressively decreasing its lumen with coils of fine wire introduced within it. From his experience it would appear that a safe and reliable method is at hand for producing partial occlusion and for aiding the development of collateral circulation.

If then, methods are available for increasing collateral circulation or if it becomes evident that the aorta can be safely occluded temporarily without obviously increased collateral circulation, one is justified in speculating further upon the possibility of excising aortic aneurysms. Should excision be possible there can be no question that one should aim ideally towards restoring blood flow through the aorta in order to prevent hypertension in cases of proximal aortic lesions and in order to circumvent intermittent claudication and other ischemic difficulties of the extremities. It is unlikely

that one could so mobilize the aorta as to permit end-to-end suture in any cases other than those of excision of a relatively short segment of the proximal portion of the descending aorta. It must be emphasized that the adult aorta is less pliable and less easily mobilized than that of the child and that rigidity and fixation of the aorta are especially prominent in cases of arterial disease. In general it would be necessary to insert a venous transplant into the defect. Experiences with vein transplantation in peripheral arteries[16] lead me to believe that vein grafts to bridge aortal defects may be feasible. It is unlikely that excision of aneurysms of the ascending aorta, the arch, or the first part of the abdominal aorta which gives rise to the important nutritive branches supplying the abdominal viscera will ever be possible. I do think, however, that it is within the realm of possibility that some day aneurysms of the descending thoracic aorta and of the distal abdominal aorta may be treated successfully by excision and restoration of continuity of the aorta. Experiments are being undertaken to test further these possibilities.

SUMMARY

A case is presented of coarctation of the aorta complicated by a saccular aneurysm distal to the coarctation and by bacteremia which may possibly have resulted from infected vegetations within the sac. The bacteremia was treated successfully by penicillin and sulfadiazine therapy and the coarctation and aneurysm were subsequently excised with repair of the aorta by end-to-end suture.

The author wishes to express his appreciation to Dr. S. H. Durlacher of the Department of Pathology, who made the microscopic studies of the excised specimen, and to various members of the Departments of Pediatrics and Radiology for their assistance.

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THE CHANGING SCENE IN AMERICAN SURGERY*

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I am honored to have been asked to give the John Chalmers Da Costa Oration. Doctor Da Costa was the first surgeon whom I met after coming to Philadelphia in 1916. I had been given a letter of introduction to him when I came East. A few days after I arrived in Philadelphia I had dinner with Doctor Da Costa at the old University Club on Walnut Street. On numerous occasions thereafter I attended his famous Wednesday afternoon clinics to which came not only students from his own school but also many from my alma mater across the Schuylkill. He was the most accomplished medical lecturer I have known. His knowledge of the history of medicine and surgery, and of current surgical literature, coupled with a rarely equalled vocabulary and form in exposition, made him a most fascinating speaker. I wonder how many of the young men in medicine and surgery have read his collected essays in "The Trials and Triumphs of the Surgeon." They might well be made required reading for medical students. He was a worthy successor to the men who so ably have filled the chairs of surgery at the Jefferson Medical College, and he occupied the Samuel D. Gross Professorship from 1910 to 1930.

Those who have read his essays know that he frequently wrote on the surgery of the past but rarely gave way to speculation on the future of the profession he so dearly loved. It was with some misgiving that I chose as my subject "The Changing Scene in American Surgery." Although we might not have agreed upon what the future has in store for us, we would, I am sure, have agreed upon an evaluation of the surgery of the past. The historian who has before him facts upon which to base his assertions is much more likely to be correct than is the crystal gazer who, with a limited knowledge of the past, attempts to be prophetic of the future.

I propose, therefore, rapidly to pass in review some of the major historical epochs in surgery before attempting to evaluate the present and future trends in surgery. It may stand us in good stead, for we are too apt to forget our debt to those who have gone before us, and it is difficult at times to reach a proper perspective of the importance of present tendencies, without some knowledge of the past.

Hippocrates, who was born in 460 B.C., can well be called the Father of Surgery, although in his lifetime all the branches of medicine were carried on by the single practitioner. He provided an excellent discussion of the lighting of an operation room, and the care of the surgeon's hands. He detailed carefully and minutely how the surgeon should work. He laid the foundation for

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* The Da Costa Oration presented on May 21, 1947, at the Philadelphia County Medical Society.

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cerebral localization, and his observations on cerebral trauma were excellent, even in the light of our present knowledge. His discussion of wounds and their treatment can be read with profit today, and his writings on fractures and dislocations were unique for nearly 1,500 years. He was the father of inductive research in medicine and he laid the foundations of physiology.

Galen, who was born nearly 600 years later, was the father of deductive research in medicine. He discovered the cranial nerves and the sympathetic nervous system and described the method by which urine flowed from the kidneys to the bladder. Yet, in spite of his keen power of observation, he frequently gave vent to riotous speculation. Charles Burr, in speaking of Galen, once said, "It was not his fault that for centuries the world accepted all his hypotheses as final," for the authority of Galen, often erroneous, persisted for nearly 1,500 years.

Vesalius, Eustachius, Fallopius and Fabricius were perhaps the four greatest anatomists of the Renaissance. These men more than any others placed morphologic anatomy on a sound basis. How tawdry are the anatomic illustrations in modern textbooks when compared to those of the great Italian School of the Renaissance.

Fabricius was the teacher and preceptor of William Harvey. He had long been interested in the vascular system and to him belongs the credit for having fired Harvey's imagination, for Fabricius had recognized the existence of valves in the veins and had noted that they were all turned toward the heart. Harvey's experiments were begun in the year of the death of William Shakespeare. A great discovery is rarely, if ever, the sole achievement of one man. Many had stood on the threshold of the discovery which Harvey finally made. Galen, in fact, had once written, "If you would kill an animal by cutting through a number of its large arteries you will find the veins becoming empty along with the arteries; now this could never occur if there were not anastomoses between them." Harvey's great work, which he wrote of in his immortal "De Mortu Cordis" was due to his use of sound experimental methods. But even Harvey did not know of the paths by which the arterial and venous systems communicate, and his discovery was not made complete until Malpighi in 1661 demonstrated by the use of microscope the existence of capillaries.

The experimental method, which was utilized sporadically, found no lasting place in surgery. The art of surgery was being practiced with a somewhat wider scope, but very few significant advances were made. Surgeons still accepted Galen's dogma that suppuration was an essential part of wound healing; they were not as yet convinced that all pus was evil.

It was not long after the time of Hippocrates that surgery became looked upon as a menial task involving simple craftsmanship. The capacity to do an operation successfully depended upon the qualities of an individual, not upon training common to all surgeons. The social and scientific standing of the "barber surgeons," who inherited this menial aspect of medicine, left much to be desired, but from this group came Richard Wiseman and Ambroise Paré.

Both were astute observers and skillful operators, and Paré, who at the age of twelve decided to become a surgeon, must always be considered as one of the greatest surgeons of all time.

John Hunter in the latter part of the eighteenth century, gave surgery the beginning of a sound foundation in pathologic anatomy. Hunter was an observer and investigator, and he was responsible for changing the spirit of surgical practice. The museum which he founded in the Royal College of Surgeons in London was the mecca of surgeons of the English speaking world until the ruthless Nazi raids on London destroyed it.

It was not until 1805 that the School of Medicine of the University of Pennsylvania deemed it expedient to separate the Chair of Surgery from that of Anatomy and Obstetrics, at which time Philip Syng Physick was appointed to the first Professorship in Surgery. Even in Edinburgh there was still a combined Chair of Surgery and Anatomy. When Physick was appointed, pain, hemorrhage and infection still impeded surgical progress. It is true that in 1799 Humphrey Davy, who had not then gained renown as a physicist, had written, "Since nitrous oxide is capable of annuling pain it might be used in surgical operations in which there is no great effusion of blood."

Can you for a moment picture the agony which was endured in the days before the introduction of general anesthesia? Hayden has given us this picture in his classic description when he says, "With a meek, imploring look and the startled air of a fawn, she is laid on the table and in spite of opiates previously administered, agonizing screams burst from her at the first cut of the scalpel. Strong men are at hand to pinion her down until the operation is completed. At length it is finished, and prostrate with pain, weak from her exertions, and bruised by the violence used, she is borne from the amphitheatre to her bed in the wards to recover from the shock by slow degrees." Such was the horror of operation in the days before Long and Morton gave to the world contributions for which we must remain eternally in their debt. Were it not for them the practice of medicine and surgery might still be little changed from that of the dark ages.

Paré had used ligatures extensively for the control of hemorrhage and Physick had experimented with the use of absorbable sutures and ligatures. By the middle of the 19th century Valentine Mott of New York had tied more large blood vessels than any other surgeon living or dead. The importance of hemostasis and methods for the control of hemorrhage became better understood.

While Physick was practicing in Philadelphia, the "living pathology" of the abdomen began to be written in Kentucky. There in what was then called the "Far West" in December, 1809, Ephraim McDowell, a pupil of Hunter, successfully performed the first ovariotomy. Thirteen years later William Beaumont, an army surgeon stationed at Michillamakinac, Michigan Territory, was called to treat Alexis St. Martin, who had been accidentally wounded by the discharge of a shot gun. St. Martin had sustained an injury of the chest wall, the left lung and diaphragm and the stomach. A year passed and Beau-

mont reported that "The injured parts were all sound and firmly cicatrized with the exception of the aperture in the stomach and the side."

Beaumont is generally regarded as the first American physiologist, but he was by act and inclination a surgeon. He was not appalled by the handicaps in knowledge which were arrayed against him. He utilized his unique patient to make lasting contributions to our knowledge of normal gastric function.

Then came the War Between the States and the best efforts of surgeons were still frustrated by infection. Of 3,117 gun shot wounds of the abdomen treated in army hospitals in the Civil War only 444 recovered, a mortality of 85.8 percent, while during World War II of those admitted to hospitals with abdominal wounds nearly 80 per cent recovered. It was not until 1881 that Kinlock of Charleston performed the first abdominal section for gunshot wound of the intestines. Those who recovered from abdominal injury in the Civil War either did not have perforation of the intestines or recovered following the formation of an abscess. It remained for Joseph Lister in 1865 to apply Pasteur's researches on fermentation to clinical surgery and to prove once and for all time that all pus is evil, that healing by primary intention, of which Paré had written more than 200 years before, could be attained.

The die was cast for a rapid expansion of surgical effort. Operations which previously could not be attempted, or, when they were, had a prohibitive mortality, began to be done not by a single surgical adventurer or genius, but by many surgeons. They were soon being done with a degree of safety that belied Baron Boyer's statement in the early nineteenth century that surgery had reached the greatest heights to which it could ever attain. The time was now ripe for a surgical approach to many hitherto unattacked lesions of the abdominal and other viscera. Many were the men who were spurred on to operate for lesions of the stomach, the gallbladder, the small and large bowel and the pelvic viscera. In Doctor Da Costa's School the names of W. W. Keen, the elder Gross, and Pancoast, and in my own Agnew, John Ashhurst and Deaver stand out in the galaxy of American surgeons who in the latter part of the last century exerted a powerful influence in the new revolution in technical surgery.

Toward the end of the nineteenth century a very great change began to take place in our medical schools. They were no longer institutes of anatomy and pathology to which were attached the clinical departments of medicine, surgery and obstetrics. Bacteriology, physiology and physiologic chemistry became independent fields of scientific effort and training. They assumed an ever increasing portion of the time of the medical student, and they played an ever increasing role in the progress of surgery.

Antiseptic surgery gave way to aseptic surgery in spite of those who believed that the older system was sufficiently good. New anesthetics began to be available to surgeons and poor methods of anesthesia gave way to better methods. Operations designed merely to alleviate temporarily the symptoms caused by certain diseases gave way to more radical and to more physiologic procedures designed to cure these disorders.

The great Billroth had issued his dictum, "Das innere medicin must mehr and mehr chirurgisch bekommen." Surgeons, following their colleagues in internal medicine, began to utilize the expanding knowledge of normal function which was made available by those working in the fundamental medical sciences, and they in turn as a result of operation in the early stages of certain diseases made available the knowledge of the abnormalities from normal function which were brought about by early disease. Prior to this time symptoms were correlated all too frequently with the findings at the autopsy table. The pathologic physiology of early disease of many viscera was poorly understood.

This was the period of the great individualists in surgery. Many of them became known throughout the world, but few of them recognized their responsibilities to their communities and to medicine, for few of them really trained many young men. The young man interested in a career in surgery still had to make his rent and board in general practice.

Progress in any art or science nearly always is dependent upon the training of the men who devote their lives to study in any special field. Surgeons in general were still trained by periods of semipreceptorship, or they were self-trained until William Halsted had the vision that such training was inadequate. As the result of his genius the resident system in surgery was born. He taught his proteges the importance of carefully studying their patients, of attempting to improve their condition prior to operation, of good anesthesia and of the gentle handling of tissues. He surrounded them during their years of training with the spirit of investigation and he encouraged them to be investigators. He forged a new method of graduate surgical training which provided increasing responsibilities and opportunities. He more than anyone else in our lifetime made surgeons realize that an operation was not successful unless the patient became well again.

In his essay on "The Trials and Triumphs of the Surgeon" Doctor Da Costa said, "It is the solemn and imperative duty of a surgeon to give able and worthy young men a chance to become surgeons. He should train them—weed out the unfit—stimulate and encourage the fit—stand by them till they can go it alone. . . . I venture to say that Professor Halsted is as proud of nothing in his distinguished career as of that splendid group of brilliant men he trained and started on the road to eminence. A surgeon who deliberately fails to train young men is guilty of a crime against humanity. A hospital management which makes a surgeon fail in this duty is criminal."

Halsted constantly stressed the fact that the ultimate aim of surgery was to restore function. He realized that to do this the surgeon must have an intimate knowledge of normal function, which is physiology, and to this his direct and indirect disciples have added physiologic chemistry and the allied basic sciences.

Such knowledge has made possible the great advance in preoperative preparation and postoperative care of our patients, achievements of the greatest importance during the past 20 or 25 years, and in the main brought about by American surgeons. Fluid and electrolyte balance, shock and its therapy,

visceral protection against noxious agents, intestinal intubation, factors influencing wound healing and many other fields have been intensively studied, and these studies have led to advances which have added immeasurably to the safety of patients who must undergo anesthetization and operation. The surgical residents, the internes and even the medical students now realize, although the lay group may not, that pre- and postoperative care is as important to the surgical patient's welfare as is the operation, and that when such care is thoughtlessly or carelessly given, it may be as catastrophic as carelessness in operative technic.

The old argument regarding the efficacy of heat or cold when applied to the abdomen of the patient with peritonitis is rarely heard today, for it is now known that neither heat nor cold applied to the abdominal wall can in any way influence the course of the infectious process. The traditional order "push fluids" of the midtwenties has been supplanted by careful orders designed to provide the individual patient with the fluid, electrolytes and other substances which that particular patient requires. It took World War II to make us realize that there is no such thing as a "blood substitute" although new and useful materials for restoring plasma volume have been found.

As the etiology of many diseases became more clearly established and the methods for cure more definitely defined there began to develop an ever-increasing group of surgical specialties. It soon became evident that patients who needed surgical care had the best chance of getting well when treated by surgeons who had been specially trained in a specific field of surgery.

The advances in surgery during the past few decades have not been concerned alone with pre- and postoperative care. The expanding knowledge of physiologic function has stimulated major extensions in technical surgery. Cushing, as a result of his knowledge of neuropathology and neurophysiology, was able to develop a specialty of the field of neurosurgery and to make it a relatively safe one. Recent additions to pulmonary, cardiac and vascular surgery have been possible because of an intimate knowledge of normal physiologic function, and the surgery of these systems has a rational and sound foundation. The surgeons who have explored, and are continuing to expand these fields could not have attained the brilliant results which they have, however great their technical skill, had they not been able to utilize the fundamental knowledge which investigators in the basic sciences have made available.

The application by surgeons of basic research to the practical problems presented by patients is frequently in the headlines, while the painstaking efforts of an imaginative scientist who made the fundamental observation is all too frequently overlooked by clinician and layman alike. Applied research is important and valuable, but it is impossible without pure research. It is an unfortunate circumstance that the importance of the latter is still too often belittled by those who do not understand its relation to the whole field of clinical medicine.

Most of the men who have contributed to the modern expansion of sur-

gical effort have been relatively young men. William Mayo in speaking of them said, "Youth without experience attacks unsolved problems which maturer age with experience avoids, and from the labors of youth comes progress." Imbued with the spirit of investigation they have perfected new methods of therapy, discovered the explanation of various clinical phenomena, improved pre- and postoperative care, and added to our knowledge of normal and pathologic physiology. They have been responsible in large part for bringing the art and science of surgery together.

The time has now come when we must take stock and determine, if we can, the direction in which surgery and surgical training shall take in the future. We have a great advantage over our predecessors, even over Doctor Da Costa, for we have had a longer period to see the benefits which come from the superior scientific training which our medical students and graduates in training are receiving. We can now be sure that there can be no substitute for planned resident training in surgery or the surgical specialties, wherein the trainee is provided with increasing opportunities for work and development, and given more and more responsibility as he demonstrates his ability.

I do not believe there is any problem in training an operator. Given a young man with dexterity it is a small accomplishment to train him to become skillful in even complicated technics. The laboratory diener who surpasses most of the students and even teachers in placing sutures is not uncommon. The young graduate who is selected for surgical training is chosen for his knowledge of science, for his broad interest in medicine, and for his ability to carry theory into practice, as well as for his manual skill. If he then receives his training in a clinic where the spirit of investigation is constantly present, where the study of the patient is made with the same thoroughness that it is made in a medical ward, he should certainly develop into a better surgeon than will the graduate who receives his training in an atmosphere of semi-empiricism and emphasis on minor technical achievements. The operator may have many brilliant operations, but the surgeon will have many well patients.

We must consolidate the experience of the past three decades in surgery and strengthen surgery below the level of specialization. I am not unmindful of all that has been done for American surgery by the American College of Surgeons and by the various academies of the surgical specialties. I am not unmindful of the benefits that have resulted from the efforts of the American Board of Surgery and the various specialty boards, but I would be remiss in my duty were I to fail to point out that a sound foundation in general surgery is the best training for any surgical specialist.

The first generation of surgical specialists was almost universally made up of general surgeons who developed particular interests in special fields and devoted more and more of their time to these fields, finally becoming known for their accomplishments in their specialties. Each of these men had his disciples whom he trained in his field, but with his own background he was able to give them of his own experience in general surgical principles.

More recently the intern has gone at once into specialized training which is too often directed by a specialist with no first-hand knowledge of general surgery. We must, I believe, strengthen the surgical specialties by providing a unity of basic surgical training. If this is not done the essential independence of the surgical specialties may become lost. As Harvey Cushing so aptly said, "They are justified only by their continued productiveness."

A year, or possibly two years, of general surgical training will provide the best possible foundation for the surgical specialist in the future. It will open to the young trainee a broader approach to his particular field, which will without doubt lead to expansions in that field. Training in general surgery may not seem to be essential to many of the older men in special fields of surgery, but it has not been many years since the older general surgeons were convinced that a background of anatomy and pathology was completely adequate for the embryo surgeon. The possession of certain specialized skills is not incompatible with a knowledge of fundamental surgical principles. The two complement each other and will inevitably lead to the advancement of knowledge in the special fields.

There are few special fields in which the trainee would not be benefited by a year or more of general surgical training. This is certainly true of urology, gynecology and orthopedic surgery. It is equally true for those who wish to enter the thoracic and cardiovascular fields. The candidates for examination in plastic surgery must now have been certified by the American Board of Surgery.

The young specialist who has been trained first in general surgery in a clinic where he has had an opportunity to do investigative work, or to come in close contact with those who are doing it, and who has acquired the ability to care for patients with all types of surgical lesions will necessarily have a broader outlook on his own work and be less limited in the technics which he brings to it. It is from such men that we may well expect leadership and the ability to expand the boundaries of knowledge in the surgical specialties in the future.

The various boards, in an effort to raise the standards of practice and to provide a gauge by which surgical specialists may be measured have attempted to standardize training. In their justifiable zeal to protect the public they have carefully and thoughtfully defined what the training for a particular field shall be. They have in many instances detailed the amount and type of basic training which candidates for certification must have. Even the number of years of training has been specified, evidently on the assumption that all training is equal in the opportunities and responsibilities presented. The experience of examiners during the past ten years has demonstrated that this is by no means true. In spite of certain defects the boards must continue to be responsible for the safeguarding of the qualifications of surgeons and of surgical specialists.

Doctor Da Costa frequently alluded to what he called "the system" in certain hospitals where "some of the staff get more than they deserve and

most get less than they need." He decried the policy which he believed to be all too common in 1915 of not appointing members of the staff purely for fitness, but of permitting personal reasons to sway the result. A period of graded training leading to certification will provide young men in the future with the necessary background which will bring recognition. If the professional staffs of our hospitals fail to recognize this, the lay boards which direct them, must provide the necessary recognition. Certification, if it is to be a hallmark of accomplishment, will signify broad education and motivated training; a full realization that the obligations of certification entails superior knowledge and skill and the ability to render distinctive service. If certification continues to signify this with increasing clarity, surgical practice will continue to improve, and the boards will play an even more important role as the custodians of general and special surgical training.

As long as medicine and surgery are further separated, they are unfortunately apt to become further distorted, for as Buckle has said, "The philosophy of any subject (that is the truth of it) is not at its center, but on the periphery where it impinges on all the other sciences." Surgery, as part of medicine, must, therefore, increasingly draw closer to the essential principles of medicine. Surgery and its specialties are not in need of new mandates—these are clear. Their function is to provide the best possible care for patients, to add to the knowledge of the field, and to train young men, not alone in the handicraft of the surgeon or the surgical specialist but in the broadest aspects of disease.

The advancement of existing knowledge and the creation of new knowledge lies in research. I am convinced that the men interested in the development of surgical laboratories, and working in them, should maintain an intimate contact with clinical surgery. These laboratories are in reality laboratories of experimental physiology and pathology as applied to surgery. The young men working in these laboratories may be concerned with work of a fundamental nature or of immediate clinical importance. It really makes little difference, for it is difficult at times to define what is and what is not fundamental research. The important thing is that such training will broaden the education of the young men engaged in it, for it will interest them in the larger aspects of disease. It will make them more critical; and it probably will enhance their eventual specialties, for they will be more apt to investigate those conditions in which they may become particularly interested. The future of the science of surgery is dependent upon such activities.

From the researches on nutrition of the early German physiologists and physiologic chemists to the present seems a long time, but there has occurred an orderly development in our knowledge of this subject which has been of incalculable value to surgeons and their patients. Starling demonstrated the importance of the plasma protein in keeping fluids in blood vessels and in their return to vessels once they have escaped. The researches of George Whipple and his associates provided fundamental observations on the regeneration of plasma protein under a wide variety of circumstances. William

Rose and his co-workers gave us new insight into the essential character of various amino acids in growth. And from these and many other fundamental studies has come new insight into the problems which many surgical patients present.

The importance of edema in conditioning respiratory complications after anesthesia and operation; the relation between hypoproteinemia and the inability of new gastrointestinal stomata to function; the part played by protein undernutrition and a vitamin C deficiency in the failure of incised wounds or decubitus ulcers to heal are now generally accepted. But protein undernutrition may result in faulty callus formation after fractures; and a similar circumstance may retard regeneration in an organ, such as the liver, which, even under only moderately favorable circumstances, possesses an irresistible urge to regenerate. It is more than probable that the two biologic factors assisting in the control of infection are dependent upon the nutritional state of the patient.

In this field, as in many others, surgery has drawn heavily upon the fundamental researches of many investigators in many fields of activity, but surgeons have made and are continuing to make contributions to our knowledge of nutrition. Robert Elman, in fact, was the first to become interested in the parenteral administration of protein derivatives, and he has stimulated an untold amount of work in this field.

The surgeons of the future will not tolerate the divorce of the hand from the brain, and the surgery of the future will not again be merely a handicraft. The surgical specialties dependent upon their productiveness will continue to multiply, but they may from time to time as Harvey Cushing prophesied "Come back to the mother tree for further suckling." In the surgery of the future the individualist will be left by the roadside, for after all surgery is part of that broader field of experimental pathology to which all the medical sciences belong.

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UTERINE ANOMALY: DUPLICATION OF UTERUS, THREE TUBES AND THREE OVARIES

Report of a Case

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Congenital anomalies of the uterus are the subject of considerable interest from an anatomic standpoint. These anomalies frequently produce serious obstetric complications as well as surgical complications.

An unusual case of anatomic anomaly of the reproductive organs warrants reporting because of failure to find any like case described in the literature. A search through the literature did not reveal any description of a case of this type. Curtis' *Monograph on Gynecology and Obstetrics,* Davis' *Monograph on Gynecology and Obstetrics,* as well as De Lee's textbook, do not describe a case of this type. In a personal communication from Dr. J. C. Masson of the Mayo Clinic, he stated that he had not heard of a similar case.

In reviewing the classifications of malformations of the uterus, particularly Jarcho's special article on the subject, it would seem that the uterus didelphys would be the classification in which to place such a case. However, this particular case does not conform to the usual description, but rather falls under one of the three differentiations suggested by Chiari:

(1) Ovaria accessoria: small split off ovarian and tubal fragments representing small appendages
(2) Ovaria bi/or pluripartita: division process of the original ovarian anlage, only two tubes
(3) True excess formation of the ovaries (extremely rare) with formation of corresponding supernumerary tubes.

The third classification of Chiari comes as near to the proper one for this case as I am able to find in the literature.

The obstetrical history in this case occurred many years before the patient came to operation, and has no bearing on the present condition.

The incident of hematometra in one side in these cases is rather common and is often the cause of surgical interference. In this case, hematometra occurred in a menopausal woman three years after the last menstrual period.

CASE HISTORY

The patient is a white female, 54 years of age, who was first seen October 11, 1946. She had been married 33 years. She was brought into the hospital by ambulance as an emergency because of severe pain in the lower right quadrant and pressure pain of the rectum. The pain had been severe for the past three or four days. The primary cause for emergency admission was a sudden rising of temperature without chill.

Physical Examination: The positive findings of a general physical examination were those of temperature of 101.4, pulse rate of 140, systolic blood pressure of 180, diastolic blood pressure of 110, rough aortic and pulmonic second sounds, occasional extrasystole, moderate cardiac hypertrophy. Abdominal examination was negative except for pain on palpation of the lower right quadrant. Pelvic examination disclosed that the vaginal vault was occluded

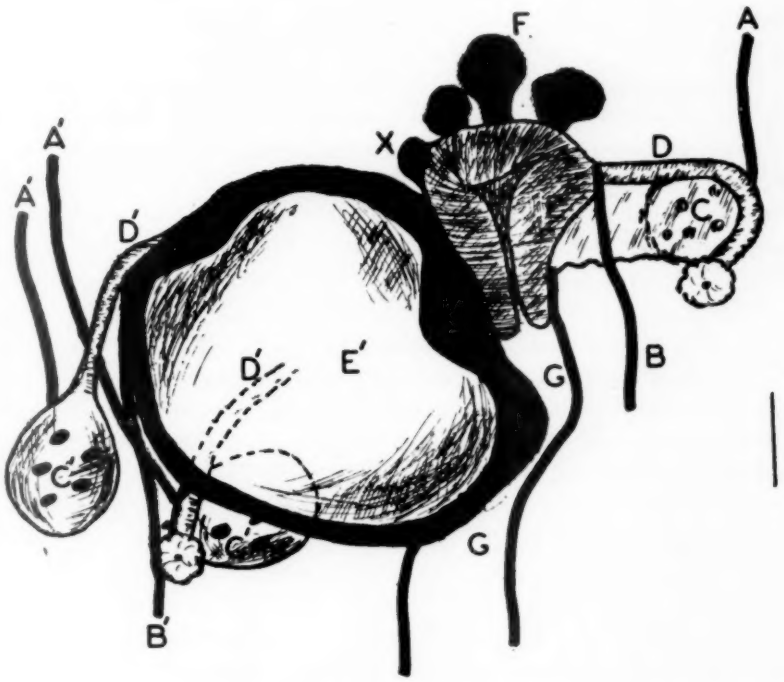

FIG. 1.—Diagrammatic drawing from the fresh specimen view from the anterior surface: A. Ovarian vessels of the left ovary of the left uterus; A'. Ovarian vessels to the ovaries of the right uterus; B. Left round ligament of the left uterus; B'. Round ligament attached to the right side of the right uterus; C. Ovary of the left uterus; C'. Ovaries of the right uterus; D. Left tube of the left uterus; X. The fundal stump of a tube arising on the right side of the left uterus, this structure having been previously removed according to the clinical history; D'. Tubes of the right uterus; E. Body of the left uterus; E'. Blood-filled cavity of the right uterus; F. Pedunculated fibroids on the left uterus; G. Vagina.

in the fornix except for a narrow slit in the left vaginal wall which would not admit the examining finger. A soft fluctuant mass was found deep in the pelvis on the right side, extending downward below the level of the cervix on the right side and bulging laterally to occlude the vaginal fornix. There was a hard nodular mass to be felt on the left side which was believed to be a uterus with fibroids. The fluctuant mass on the right was believed to be either a uterine cavity full of fluid or a ligamentous ovarian cyst. On speculum examination the cervix could not be seen but a large bulging mass could be seen in the upper vault of the vagina, excluding the entire fornix except for a

small slit on the left side between the left vaginal wall and the bulging surface of the right vaginal wall. Through this opening a small probe could be passed and a hard nodular structure was felt which was assumed to be a small cervix.

The history of past attacks was as follows:

Menstruation began at the age of 13 and was markedly irregular from the onset to the sixteenth year of age, with severe headaches attending each period. At the age of 16 she had her first attack of lower right abdominal pain. She also had a pain low in the rectal region. This is the same type of painful distress that she complained of upon admission. She was operated upon at the age of 16 for what was presumed to be appendicitis. The operation was performed in a small mining hospital, and there are no records obtainable. So far as the patient can recall, the appendix was removed and she was told there was a small tumor removed which was called a fibroid, and that one ovary was also removed with an adjoining tube. After recovering from this operation her menstrual periods became regular, occurring every 28 days and lasting from three to four days. This regularity continued until the age of 31 at which time, though the menstrual periods occurred regularly, she had a recurrent attack of pelvic distress and was again operated upon. An exploratory operation at that time revealed a pregnancy in what was described to her as one uterus, and also revealed a tumor mass on the right side; also the presence of a fibroid growing from the uterus on the left side. The fibroid tumor was removed at this operation. No other procedure was done so far as the patient can recall. This operative record is also unobtainable. However, she was told that she was pregnant; and seven days postoperative she expelled a fetus which was said to be of five months' gestation, and which could be identified as a male. Following this second operation her periods were regular and for several years she had no distress. Prior to her menopausal change her periods were associated with severe headache. Periods stopped abruptly at the age of 51, approximately three years before she was examined at this clinic.

On further questioning after the operation, the patient stated that one of the doctors who operated on her the first time told her she had four ovaries and four tubes. This statement is borne out by our subsequent findings.

In addition to her pelvic history, patient gave a history of food dyscrasia, particularly with fats, associated with gas and belching, indicating some gallbladder disturbance.

Laboratory findings on admission were as follows:

Blood count showed a hemoglobin of 84 per cent; 4,370,000 red cells; 8,300 white cells, 76 polys, 8 stabs and 24 lymphs. Clotting time was 12 minutes. The non-protein nitrogen content was 40 mgm. per 100 cc. of blood. The Kahn and Mazzini were both negative. Voided specimen was cloudy, pH was 6, specific gravity 1.005, albumin I on the basis of IV, and there was no sugar.

At the time of admission to the hospital the patient's condition was acute. The low pelvic pain which had been present for several days had become severe and was constant in character. It was decided that exploratory surgery

was advisable. She was therefore operated October 12, 1946, with a tentative diagnosis of pelvic mass on the right side which could either be a uterus with retained menses or an interligamentous ovarian cyst; and fibroids of the uterus. Also, malformation of the pelvic organs.

Operative Procedure. Through a midline incision excising the old scar the abdomen was opened and after proceeding with the dissection of adhesions and freeing the pelvic organs from the bowel and omentum the following anatomical structures could be identified: there was a uterus on the left containing fibroids. There was an ovary far to the left, normal in appearance, and a chronic infected tube properly located. On the right side of this uterus there was a small nodular mass that appeared to be the site of a previously existing tube which had been removed. Many adhesions of the omentum were dissected from this area. There could be no ovary identified lateral to the fundus of the uterus containing fibroids. About the midline a cystic ovary could be identified and following the tube toward its attachment we found a large mass —the fluctuant soft mass felt on examination. The tube entered this mass and anterior to the tube a round ligament was identified. This was the right uterus. The cavity of this uterus was full of brown material which was retained menses. There was also a round ligament identified from the left uterus and from its fundal insertion it could be traced to the left inguinal insertion. On freeing the right uterus as a fluctuant mass another ovary and tube were found attached in the proper relations which had hitherto been hidden because of their position deep in the cul de sac. General abdominal exploration revealed fiddle-string adhesions between the diaphragm and the liver which suggested the possibility of Neisserian infection at a previous time. The gallbladder was bound in a mass of fat and omentum which was adhered to the liver edge. No stones could be palpated. The head of the cecum was bound down in such a mass of adhesions that it was deemed best not to disturb it to try and determine whether or not the appendix had been previously removed. On removing the pelvic structures an ovarian vessel was identified supplying blood to the left ovary of the left uterus. We were unable to identify a separate vessel which might have supplied the right ovary of the left uterus. There were two distinct ovarian vessel groups supplying each of the two ovaries of the right uterus. These were separated by a distance of one and one-half inch. They were separately ligated. The ureter on the right side could not be identified. The ureter on the left side was identified. It was necessary to remove the left uterus and open the vaginal vault on the left side to approach and remove the very low-lying right uterus. A pan-hysterectomy was done, metaphen sponge was placed in the vagina, the vaginal mucous membrane was closed by opposing the mucous membrane with interrupted #1 chromic sutures placed in the muscularis. The cardinal ligaments were sutured to the cervical stump on either side, uterosacrals were caught in a circular suture which contained each uterosacral ligament, the two round ligaments which were identified, and the stump of the cervix. There was a very large dead space left on the right side which was dry when the peri-

toneum was closed. It was possible to find sufficient peritoneum to cover all the raw surfaces. The abdomen was closed without drainage.

On examining the pathologic specimen postoperatively from a surgical standpoint, it appeared that there was a uterus on the left side with an attached tube and ovary and a round ligament attachment. There was a large fibroid in the fundus of the uterus and a large fibroid in the region of the cervix and three small pedunculated fibroids on the outside of the uterus posteriorly. There was an area on the right side of the left uterus which appeared like the site of a tube that had been attached at sometime. The uterus on the right side was thin-walled; there was a large cavity containing a thick brownish-green material which was retained menstruum. This uterus had a tube and ovary on the right horn and a tube and ovary on the left horn. The cervix of the right uterus was completely closed at the internal os. There was a continuous fibrous band from the internal os downward about one inch. The cervix of the left uterus had a very fine narrow canal which would open into the vagina. We therefore had a specimen consisting of three ovaries, three tubes and two uteri.

The report of the pathologist is as follows:

"Congenital malformation of the reproductive organs: Two uteri, one with two tubes and two ovaries; the other with one tube and one ovary;

Multiple fibromyomata, subserous and intramural, of one uterus;

Atrophic ovaries attached to the myomatous uterus;

Hematometrium and wall atrophy of the second uterus;

Hematosalpinx and endometrioma of the ovary attached to the second uterus;

Massive old adhesions around the second uterus, tube and ovary."

The pathology was reported by Dr. Seigfried Werthammer, Huntington, West Virginia.

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THE SELECTION OF PATIENTS FOR THORACOLUMBAR SYMPATHECTOMY

Description of a Set of Rules for the Elimination of Failures and Fatalities

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In a series of 375 patients undergoing thoracolumbar sympathectomy we have had 38 fatalities either in the hospital or within 6 months of the operation. In an attempt to avoid future deaths we have analyzed the status of this group of 38 patients and also that of the remaining 337 patients and have arrived inductively at a set of rules which would eliminate all but 2.5 per cent of the mortality. The figure 2.5 per cent is reasonably low in view of the fact that each patient undergoes two major operative procedures and in this group of patients, many are recognized as poor risks.

Following the lead of Keith, Wagener and Barker[1] who graded the eye-grounds of hypertensive patients from 0 to 4 plus we have utilized a similar method of grading the cerebral, cardiac and renal status of each patient. In order to do this satisfactorily one needs in addition to a careful history and physical examination the following studies: fundal examination, electrocardiography, six foot heart plate, a concentration test (pitressin or Mosenthal), urea clearance, blood urea nitrogen, non-protein nitrogen, creatinine and urinalysis. Intravenous urography was applied routinely in the work-up of the first 150 patients until one death and two marked reactions associated with the injection of the dye caused us to abandon it unless there was a significant indication for its use. A complete blood count, sodium amytal test (nine gr. divided into three hourly doses) and a basal metabolism are desirable but not emphasized in evaluating the patient's status for sympathectomy. A high basal metabolic rate associated with the hypertension makes one suspicious of a pheochromocytoma.[2] The sodium amytal test is a good indication of the probable postoperative blood pressure result but is not absolutely accurate as a prognosticator in any given case.[3] The Etamon test (tetraethylammonium chloride) has been studied by Dr. C. A. Poindexter and Doctor Tamagna and correlated with the sodium amytal test in more than 30 patients. In approximately 75 per cent of the patients the two tests were correlated exactly, while in the remaining 25 per cent there was a variation in the Etamon test in both directions from the sodium amytal test. No post-operative evaluation of its possible prognostic usefulness has been made.

In Table I we have outlined the definitions which serve to evaluate the degree of damage present in each of the four important organs, brain, eye, heart and kidney, as a result of the hypertensive state. In Table II a group of rules are set forth for the selection of patients based on the definitions as described.

In the preoperative evaluation of the status of an organ problems such as the following may arise: the renal concentration test may be only 1,013 whereas the urea clearance may be 85 per cent of normal. In that case we have one test placing the kidney in a three plus category and the other in a one

TABLE I.—*Definitions*

In general in any system
- 0: Normal
- 1: +Slight or mild changes
- 2: +Moderate changes
- 3: +Moderate changes
- 4: +Advanced or marked changes

Cerebral
- 0: No symptoms or signs
- 1: +Headaches and/or dizziness and nervousness
- 2: +Above plus nosebleeds and/or occipital headaches
- 3: +Above plus paresthesias
- 4: +Stroke or encephalopathy or confusion

Eyes
- 0: Normal
- 1: +Arteriolar narrowing
- 2: +Above and arterio-venous nicking
- 3: +Above plus hemorrhages and exudates
- 4: +Above plus papilledema

Cardiac
- 0: No symptoms or signs
- 1: +Slight symptoms and/or slight cardiac enlargement and slight E. K. G. changes
- 2: +Moderate symptoms and/or moderate X-ray enlargement and moderate E. K. G. changes
- 3: +Marked symptoms and/or marked enlargement and marked E. K. G. changes
- 4: +Coronary occlusion or congestive heart failure

Renal
- 0: Normal
- 1: +Nocturia, but concentration 1.024 or more and urea clearance 75% or more
- 2: +Urea clearance 40–75%, concentration 1.015–1.023
- 3: +Urea clearance less than 40%, concentrate less than 1.015, normal blood chemistry
- 4: +Persistent elevation of N. P. N. to 45 mgms. or more and B. U. N. to 25 mgms. or more

TABLE II.—*Rules*

A. *Contraindications to thoracolumbar sympathectomy*
1. 4+Renal
2. 4+Cardiac in which congestive heart failure is unremitting or if coronary occlusion is within 3 months
3. 4+Cerebral if confusion exists or if a stroke within 3 months
4. If there are two 4+ other than eyes
5. If total count equals 11 or more pluses

B. *Indications for thoracolumbar sympathectomy*
1. All cases are operable in which there is no contraindication rule
2. From the viewpoint of minimal involvement: Operation is probably advisable in patients with persistent hypertension associated with definite though minimal objective changes in any one of the four systems

plus. We, therefore, have averaged the two and considered the degree of damage as two plus. Similarly a patient may have excellent cardiac function, *i.e.*, no dyspnea or angina in climbing three flights of steps and yet the electrocardiogram and chest plate may show moderate (two plus) changes. It is a matter of judgment whether the patient should be classified as a two plus or a one plus cardiac.

A system such as this one, not based on mathematical data, must be interpreted in the light of clinical judgment, and implies that the surgeon operating on these patients has had moderate experience with the technic of thoracolumbar sympathectomy. The rules have aided us considerably, however, since their formulation and may be of help to internists and surgeons interested in the surgical treatment of hypertension.

A few comments on special aspects of the problem of selecting patients for surgical therapy should be added:

1. Generalized arteriosclerosis of a significant degree is a bad sign and should exclude a case of borderline acceptability.

2. Age is not a factor in selection. In our patients 50 years or older (two in the sixties) the same percentage of worthwhile results (*i.e.*, 67 per cent) was noted as in the entire group operated upon.[4]

If, in this series of 375 patients, the contraindication rules outlined had been applied, then 25 patients who have done well would have been refused operation. Among the remaining 350 patients there were 38 deaths either in the hospital or within six months post-operatively, 30 of these would have been eliminated by the rules outlined in this paper, leaving eight deaths in a total of 320 patients, a mortality of 2.5 per cent.

The problem of determining which of the 312 patients, who were within the limits of the contraindication rules and who lived through the operative procedures, would derive excellent results and which ones would derive only fair results or no benefit, is a most difficult one. Smithwick has emphasized that females Type 1 (narrow) pulse pressure obtain the best results and that males with Type 3 (wide) pulse pressure do poorly. We have seen many exceptions to this rule and as yet have found no system which will work in the majority of the cases. Therefore at present we adhere to the policy of advising an extensive thoracolumbar sympathectomy on all patients who have a sustained hypertension, unresponsive to the usual medical management including the rice diet, and who have definite though mild objective changes in one or more of the four major organs discussed above.

It should be emphasized again that the set of rules discussed in this paper is not a substitute for clinical judgment and individual consideration of each patient but rather serves as a useful adjunct and as a working basis for the elimination of poor risk cases.

SUMMARY

On the basis of an experience with 375 hypertensive patients undergoing thoracolumbar sympathectomy we have arrived inductively at a set of rules which would have eliminated 30 of the 38 deaths which occurred in this series either in the hospital or within six months postoperatively. A discussion of the rules and the definitions on which they are based has been given.

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CONSTRICTIVE PERICARDITIS WITH TUBERCULOUS INTRAPERICARDIAL ABSCESS TREATED BY STREPTOMYCIN*

Report of a Case

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THE ROLE OF TUBERCLE BACILLUS as a causative agent for constrictive pericarditis is recognized by recent investigators.[1, 2, 3] Pleural effusion, a common finding in patients with constrictive pericarditis, is thought to be part of a polyserositis rather than a sequela of the constrictive pericarditis.[3]

Sellors[3] reports that pockets of fluid or inspissated debris are often present in the pericardium. A survey of the available literature,[1-12] however, reveals no report of a definite intrapericardial abscess from which acid-fast organisms were recovered, accompanying the constrictive process. No cases have been reported in which streptomycin was used either pre- or postoperatively.

Our case of constrictive pericardial involvement followed the typical course with bilateral pleural effusion and pericardial effusion followed by slowly developing signs of venous obstruction. At operation a constrictive pericarditis with an intrapericardial abscess was found. After pericardectomy streptomycin therapy was administered and the patient recovered without a draining sinus in spite of the proven presence of tubercle bacillus in the intrapericardial pus.

CASE HISTORY

L. S., a 20 year old Negro male, was admitted to the Veterans Administration Hospital at Oteen, North Carolina, on September 9, 1944. He was inducted in December, 1941. In December, 1943, while in England, he had what was termed atypical pneumonia and was off duty for a period of six weeks. Roentgen-ray at this time was said to have shown a normal heart shadow. Six weeks later, in May, 1944, he became ill with chest pain, cough, shortness of breath and fever. Roentgen-ray examination in an Army hospital revealed an enlarged cardiac shadow. The tentative diagnosis at this time was pericarditis, probably of rheumatic origin. Soon after admission the pericardium was tapped and 80 cc. of straw-colored fluid was obtained. Cultures of this fluid were negative for acid-fast and pyogenic organisms. Fever, however, persisted. He stated that fluid was removed subsequently, twice from the pericardium and once from the left pleural cavity. In July, 1944 he was returned to a United States Army Hospital where he was found to have a residual left pleural effusion and a recent effusion on the right.

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A right thoracentesis, however, was unproductive. His temperature ranged up to 104°F. and he was kept on salicylates. The sputum was repeatedly negative for tubercle bacillus, but the tuberculin test was strongly positive. He was then transferred to the Oteen Hospital where a diagnosis of polyserositis with a pericarditis was made. The temperature ranged from 99 to 101° F. from September 9, 1944, to January 9, 1945, when it approached normal and remained there with occasional minor elevations.

During his entire period at Oteen, numerous concentrated sputum studies were negative for tubercle bacilli, as were cultures and guinea pig inoculation made from concentrated sputum and from gastric washings. On admission, fluid was present in both right and left pleural cavities which, when removed by thoracentesis, proved to be negative for tubercle bacilli and any other organisms. The urine showed a trace of albumin on numerous occasions, with an occasional white blood cell and occasional granular casts. The liver was enlarged two fingerbreaths below the costal margin. There was moderate ascites. Abdominal paracentesis on September 12, 1944, yielded 80 cc. of straw-colored fluid which was negative on culture for any pathological organisms. Dyspnea became progressively more severe. Edema of the ankles was present. Roentgen-ray showed that the borders of the heart were obscured by fluid collection, but the heart shadow was considered widened. By February 28, 1946, the heart was reported to be globular in type and not unduly enlarged. On June 5, 1946, the roentgenologist reported the heart to be normal in size, although it was displaced a little to the right. There was obliteration of the costophrenic angle bilaterally due to the residuals of pleuritis.

The venous pressure on July 2, 1945, was 160 mm. of water. The circulation time was 13 seconds from arm to tongue. By June 5, 1946, the venous pressure had risen to 240 mm. and with pressure over the liver was elevated to 270 mm. Circulation time, however, was only 11 seconds. There was marked dilatation of the veins of the neck and upper chest. A diagnosis of constrictive pericarditis was made and a pericardectomy performed June 6, 1946. The pericardium was thick and adherent but a line of cleavage was rather easily identified and the pericardium removed by blunt and sharp dissection, beginning with the left ventricle. In the upper lateral and posterior portions of the pericardium covering the left ventricle was a dense area of adhesions which, when separated by sharp dissection, opened into a pocket containing approximately three ounces of thick, creamy pus. This pus was sent to the laboratory and was positive on smear for acid-fast bacilli. The excision of the pericardium was then carried over to the right ventricle. Following the excision of the pericardium the cardiac impulse seemed to be greatly improved. One gram of streptomycin was dissolved in saline and was instilled into the remains of the pericardial sac. The wound was closed tightly without drainage.

The pathologist reported the pericardium to be 0.5 cm. in thickness. Microsection revealed thickened and hyalinized strands of connective tissue with attached granulation tissue consisting of large and small mononuclear cells, red cells, fibroblasts and new formed vessels. A pathologic diagnosis of chronic granuloma was made.

The pathologic reports in this case tend to explain the confusion which is apparent in the literature as to the role of tuberculosis in the etiology of constrictive pericarditis. Here we had definite proof of the presence of acid-fast bacilli in the pus obtained from the intrapericadial abscess. Microscopic examination of the tissue, however, did not give a clear-cut picture of tuberculosis and the pathologist was unable to make a diagnosis other than chronic granuloma.

The day following the operation the venous pressure which had been 240 mm. of water preoperatively was found to be 132 mm. Circulation time was 13 seconds. The venous pressure gradually rose, however, until on August 10, 1946, it had again reached 220 mm. Circulation time was 16 seconds at that time. Following the operation, streptomycin, 2 grams daily, was given until July 22, 1946. There was a small amount of drainage of a serosanguineous nature for about one month after the operation but all dressings were discarded on July 3, 1946, and the wound remained dry. Clinically the

patient improved markedly and was discharged from the hospital on November 7, 1946, without ankle edema.

On July 10, 1947, thirteen months after the pericardectomy, the patient stated in a letter that he was not short of breath and could walk ten blocks without difficulty. He reported that his ankles were not swollen and that his abdomen was not enlarged. If he climbed a flight of stairs at a rapid rate, however, he became short of breath. Circulation time and venous pressure studies were not available at this time.

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"FUNCTIONAL" SUBCLAVIAN ARTERIAL MURMUR: POSSIBLE RELATION TO SCALENUS ANTICUS SYNDROME, COSTOCLAVICULAR COMPRESSION, OR THE NEUROVASCULAR SYNDROME OF WRIGHT

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The purpose of this paper is to present cases of apparently normal individuals in whom was found, on routine pre- and re-employment examination, a group of signs—and no symptoms—referable to the subclavian artery of one or both sides; and to speculate, in the light of certain findings, (particularly the evidences of arterial obstruction), on the possible origin and significance of these signs.

INTRODUCTION

Consulting standard texts on physical diagnosis, the following comments are found: "Constriction of larger vessels will produce murmurs. An example is the systolic murmur sometimes heard a short distance below the clavicle due to the narrowing of the subclavian in some part of its course";[1] "In coarctation of the aorta, there is a soft systolic murmur over the innominate, carotid and subclavian arteries. This murmur is often heard at the angle of the left scapula";[2] and "Subclavian (functional) murmurs, heard best in inspiration, are most common in men and are modified by position of the arm. Subclavian murmurs possess no pathologic significance; they are generally due to constriction of the artery between the clavicle and first rib, although . . . may be . . . fibroid disease of the pleura. Landis found of 31 cases . . . 20 associated with pulmonary tuberculosis. The fact that it is heard more on the left . . . suggests anatomical variation."[3] Perusal of the Index Medicus for the years 1940 through 1945 under the titles "arteries," "murmurs" (nothing listed), "muscles, scalene," and "pulse" reveal nothing by title that might bear directly on the explanation of the murmur in question. References to the signs of subclavian or axillary arterial obstruction are cited as they apply to the discussion (v.i.).

CLINICAL MATERIAL

These patients, representing 21 of 2619 applicants for position, or 0.8 per cent, came to attention solely because of a systolic murmur over the subclavian artery (or arteries) which was discovered incidental to the routine examination of the chest. The first 11 of this group were passed over with no special examination, the condition being assigned in the examiner's mind to the class of functional murmurs (see Table I). Later, however, more detailed examination of ten persons was performed with the results that will be brought out later in this communication.

Twenty of the 21 persons were females (ratio of total females to males was only 7:1), the average age was 21 years, and the murmur predominated

on the left, appearing on that side alone 11 times, on the right side alone only once, bilaterally five times, and in four cases the laterality was not noted. The personnel examined was entirely of Mexican extraction, approximately two thirds of whom were born in the United States. As far as could be determined, the cases demonstrating a murmur had nothing in common in their past histories, and as usual in this age group, were generally negative on physical examination. Exceptions to this are noted in full. Special care was taken to determine that this was not in any case an "underground" murmur from a valvular heart disease and no patient had signs or symptoms suggestive of heart disease. A routine Mazzini test was negative in all cases; a routine

TABLE I.—*11 Cases: No details as to B. P.*

Name	Age	Sex	Side of Murmur: Right	Side of Murmur: Left	Remarks:
EG	20	F	..	*	Fluoroscopy negative
AG	21	F	..	*	Fluoroscopy negative
ME	18	F	X	X	Fluoroscopy negative
SM	23	F	..	X	Fluoroscopy negative
RG	20	F	..	X	Fluoroscopy negative
ME	19	F	X	..	Fluoroscopy negative. Present on inspiration only
MGP	38	M	..	X	Fluor: Undiagnosed pulsation of abnormal amplitude, left border of heart above ventricle—? No cardiac murmur.
VD	21	F	..	X	Fluoroscopy negative
FT	..	F	..	*	Fluoroscopy negative
TT	..	F	..	X	Fluoroscopy negative
MBV	..	F	..	*	Murmur on inspiration only
Average 10 F					
11	22.5	1 M	2	6	

* Presence of murmur recorded, but laterality not noted.

Roentgen-ray and/or fluoroscopy of the chest was negative in all except one. This worker, Case 3, has had apical tuberculosis, minimal, and now inactive; this finding is of four and one-half years duration at the time of this writing and the patient has been asymptomatic during that period, maintaining normal weight and working eight hours per day at a moving belt. It will be noted that this one case of tuberculosis is on the side opposite that of the murmur. The incidence of active tuberculosis in the total group of employees examined was 1.2 per cent, but another 2 per cent are under periodic surveillance as "suspicious" cases and probably represent healed lesions. This incidence of relation of the murmur to tuberculosis is insignificant compared to the representation, above cited, by Landis.[3]

It is important to add that, although no symptoms were presented, neurologic examination was nevertheless done in all cases (*i. e.*, pin prick and cotton), and no instance of cervical plexus interruption was found.

DESCRIPTION OF THE MURMUR

The murmur itself, as may be correctly inferred from the fact that it was found on a brief routine examination, was constant for the individual, fairly

loud and definite. It would not always have been heard were it not routine with the examiner to have the patient place the hands on the hips for the auscultation of the axillae, because the murmur is sometimes heard only on elevation occurring with this position. On other occasions the murmur was heard in the relaxed sitting position, but not heard in the recumbent position. When heard in the relaxed sitting position, or in the hands-on-hips position, the murmur was always accentuated by deep inspiration or gradual abduction of the arm. The greater the abduction, the louder the murmur, up to abduction of 135 to 150 degrees (Nos. 1, 2, 5, 7, 9, and 10—see Table II), when the murmur disappeared altogether because blood flow to the arm as ascertained by radial pulse and blood pressure* had been obliterated. In four cases (Nos. 2, 3, 7, and 9), the murmur had been heard from four to six years previous to its more detailed investigation. These patients had neither developed symptoms nor demonstrated any appreciable change in the nature of the murmur.

The accentuation of the murmur upon elevation of the arm, suggested the performance of a group of somewhat unorthodox tests to supply information for functional analysis. With the patient seated, blood pressure was taken in one arm, at first relaxed and then voluntarily elevated above the head to 180 degrees abduction. This was then repeated on the opposite side. Following these readings, a blood pressure was taken on one thigh, using the popliteal artery as the point of auscultation. (This latter determination was made to further exclude in an objective manner possible cases of coarctation, and in no case was the blood pressure in the lower extremities lower than that of the upper.) Now, in order to correlate the various readings for easy interpretation, a mean blood pressure was calculated for each arm, averaging the systolic readings relaxed ("down" in the chart) with that in extreme abduction ("up" in the chart) and similarly for the diastolic, "down" and "up." By comparing these figures for the affected and unaffected sides, probably the most information concerning the blood flow to the arm is obtained—(Table II). Also, pulse pressure difference ("P. P. D.") between the arm at rest and elevated was calculated for each side. The figures resulting from this calculation are less informative because, although an affected side may have had a lower mean blood pressure, the pulse pressure difference may actually appear to be less in the affected arm by reason of the fact that some of these vessels showed a moderate difference in blood pressure when comparing the two at rest. The typical cases, which showed a marked fall in pulse pressure on elevation of the affected side, did so by reason of the fact that the resting blood pressures were the same in both arms, or nearly so, (Cases 1, 2, 3, 6, 7, 8, and 10); the atypical cases with a difference in blood pressures in a resting position showed an equal or lesser fall of the P. P. D. of the affected side, as compared with the normal. The bilateral cases can be compared only

* Palpation of brachial pulses too weak to be propagated to the radial artery were considered a function of the collateral circulation.

with the normal sides of unilateral cases.** Reference to Table II will show that in the presence of bilateral murmurs, the obstruction to blood flow is not usually the same on the two sides, and was generally greater on the left. In

TABLE II.

Case No.	Age	(All ♀ or ♀ Females) Side of Lesion Rt.	L.	Blood Pressures—mm Hg Right Down	Up	Mean B.P.	Left Down	Up	Mean B.P.	"P.P.D"† Rt.	Left	Duration (yrs.)	Remarks
1	17	X	X	120	None	60	110	None	55	40	40	0	X-ray neg. except horizontal and externally flared first ribs. Murmur on right only. Both pulses cease on elevation and cyanosis of both hands and poor capillary return (color).
				80		40	70		35				
2	20	X	X	140	105	122	122	98	110	15	8	6	Left radial ceases on elevation. Fluor. negative. X-ray negative.
				75	55	65	60	44	52				
3	39	..	X	138	115	126	145	84	115	8	26	4½	Tbc. rt. apex, minimal, quest. activity. Clinically o. k.
				80	65	73	90	55	73				
4	16	X	X	135	None	63	105	70*	88	60	?	0	Murmur heard only when head is rotated. X-ray: Pin point calcification left supra clavicular fossa.
				75		37	70	?	?				
5	22	..	X	115	85	100	90	70	80	20	5	0	Murmur on inspiration only. X-ray negative. On inspiration, no reading in left arm, elevated: PPD 35, under these circumstances.
				80	70	75	60	45	53				
6	26	..	X	128	90	109	128	75*	101	23	?	0	X-ray negative.
				75	60	68	70	?	?				
7	22	..	X	110	70	90	110	0	55	20	40	4	No pulse on left, elevated above 135 deg.
				70	50	60	70		35				
8	16	X	X	140	90	115	140	98	119	30	12	0	Slight thyroid enlargement but no bruit. X-ray negative. Bilateral murmur sitting position only.
				90	70	80	95	65	80				
9	26	..	X	125	85	105	90	0	45	30	20	5	Murmur on inspiration only and elevation of arm. Blanching and tingling of left hand and forearm on elevation. X-ray negative.
				80	70	75	70		35				
10	16	..	X	115	90	103	115	55*	85	—5	?	0	No brachial sound on elevation. Murmur only on elevation left arm. Complaint: tingling of hands on elevation only. X-ray negative.
				80	50	65	80	?	?				

† "PPD" is the remainder of the pulse pressure of "Down" minus the pulse pressure of "Up."
* Palpatory. No sound heard over brachial. No significant oscillation of brachial.

** The average fall of pressure on the normal sides were: systolic 32.8, diastolic 16.7, pulse pressure 16.1. On the abnormal sides: systolic 66.7, diastolic 50.8, pulse pressure 15.9. PPD, therefore, is significant only when comparing the two sides of a single case. (All pressures are here and subsequently expressed in millimeters of mercury.)

some cases palpatory blood pressures were taken to represent the systolic pressure when no auscultation was possible over the brachial artery. In Cases 1, 2, and 7 auscultation was possible, but no radial pulse, nevertheless, could be found. This observation is explicable on the basis of peripheral arterial spasm: these patients complained of tingling or coldness on the affected side, and demonstrated varying degrees of ischemia of the elevated hand in comparison with the opposite side.

CASE REPORTS

From the information derived from the reports of previous investigators, the relation of the constriction of the subclavian-axillary segment to either scalenus anticus syndrome, costoclavicular mechanism,[5] or the common anatomic arrangement of the coracoid process and pectoralis minor muscle and their relation to the vessels[4]—an attempt was made to pick out those cases which fell into each class on the basis of the maneuver which caused the obstruction, and in these cases the murmur. The following cases were all females, and to concur with one of Wright's statements, most of these individuals were not muscular and yet had markedly reduced blood pressures and pulse pressures due simply to hyperabduction. In six of the patients, the peripheral pulses disappeared.

In this series no case was observed which was thought to be a scalenus syndrome, and only one was thought to derive from costoclavicular compression, namely Case 1. The remainder followed the criteria of the obstruction of the artery, in varying degrees, on hyperabduction of the arm and, therefore, fell into the new anatomic classification brought out by Wright in the discussion of the neurovascular syndrome.

Case 1.—B. T., 17 years, on routine examination was found to have the murmur on the right side only, just below the clavicle, but in comparing blood pressures, it was noticed that the left arm consistently gave 110/70, or less than that of the side of the lesion which was 120/80. On hyperabduction, the brachial and radial pulses disappeared bilaterally and there was cyanosis of both hands in a few seconds. Roentgen-ray demonstrated markedly flared first ribs, the upper surfaces being almost horizontal.

This was the only case which showed obstruction to the venous in excess of the arterial supply (collaterals)—if one is willing to interpret cyanosis as indicating this condition.

The relation of obliteration of peripheral pulse to deep inspiration as being of costoclavicular mechanism is strongly supported by the findings in the case which Schumacher[7] operated under the diagnosis of subclavian aneurysm. Correlation is not conclusive because the author reports that the roentgenograms were suggestive of, but not definite for, costoclavicular compression and makes no note of having moved the arm to a position of hyperabduction when the artery was under direct observation. The murmur was present in the relaxed standing or sitting position, however, just as in this case.

The remainder of the cases were characteristic of the type of obstruction due to hyperabduction alone. It is my impression that had Wright made

routine blood pressure determination on his "normals" that a definite difference in resting blood pressures (between the two sides) would have been detected in some. This conclusion is based on the assumption that the following cases are examples of "asymptomatic neurovascular syndrome"—for want of a better term—and that some as yet undefined mechanism operates to obstruct the artery in these cases even before the exercises in abduction are begun. This is supported by the presence of the murmur in some cases in a relaxed sitting position.

Case 2.—A. M., age 20, bilateral murmur (6 yrs. duration) had a difference in resting blood pressures (140/75 on the right; 122/60 on the left) demonstrated reductions of the blood pressures and pulse pressures on hyperabduction: right 105/55, left 98/44. The left radial pulse was not palpable on elevation, but the blood pressure was obtained repeatedly, and with good oscillation of the mercury column.

Case 3.—R. F., age 39, is the patient previously described who had a healed minimum tuberculosis of the right apex and in whom a murmur appeared on the left only, had resting blood pressures of 138/80 on the right and 145/90 on the left, but subsequently showed obstruction of such degree that the right blood pressure fell only to 116/65 on hyperabduction whereas the left fell to 84/55. She had no symptoms referable to the left arm during the known duration of the murmur, which was for 4.5 years.

Case 4.—A. B., age 16, murmur present bilaterally but only when head was rotated to the corresponding side, suggesting a possible involvement of the scalenus. This was refuted by the observation that with hyperabduction alone no blood pressure was obtainable in the right arm and a palpable blood pressure of 70 was obtained on the left. It was also noted, on the left, that although the pulse could be felt in the cubital fossa, no oscillation of the mercury column was observed when the cuff was deflated from the resting systolic pressure to zero. This failure of the mercury column to oscillate was present in but 3 of 10 cases.

Case 5.—G. A., 22, left murmur appeared on inspiration only. There was a difference in the resting blood pressures: right 115/80, left 90/60. On hyperabduction pressures fell to: right, 85/70, left 70/45. Hyperabduction plus deep inspiration obliterated pulse in the left arm. The additional affects of inspiration and elevation of the arm suggest that the obstruction to this left subclavian artery could well be a combination of costoclavicular and coracoid process obstruction.

Case 6.—Z. P., age 26, murmur on left only. Resting blood pressures: right 109/68, left 128/75, fell to: right 90/60 and left, no auscultatory point but could palpate pulse at 75 and again observed the phenomenon of complete lack of oscillation of the mercury column.

Case 7.—C. H., age 22, murmur on left only. Resting blood pressures equal—110/70 fell to: right 70/50 and neither sound nor pulse could be obtained on the left elevated above 130 degree abduction. There has been no change in this murmur over a period of 4 years.

Case 8.—E. H., age 16, had a bilateral murmur in the relaxed sitting position, and a slightly enlarged thyroid. Question of substernal thyroid or other mass was subsequently ruled out by roentgen-ray examination and the murmurs were not bruits characteristic of a vascular thyroid. Resting blood pressures were: right 140/90, left 140/95; on hyperabduction these fell to right 90/70, left 98/65.

Case 9.—O. F., age 26, left murmur in hands-on-hips position only. There was a difference in the resting blood pressures: right 125/80, left 105/75 and on hyperabduction

the right fell to 85/70 and the left was neither audible nor palpable. In addition, the patient complained of tingling of the left hand and forearm on hyperabduction and the left hand was noticed to be pale in contrast with the right.

Case 10.—M. M., age 16, had the murmur in hands-on-hips position only. Resting blood pressures were equal—115/80. On hyperabduction the right was 90/50; on the left the phenomenon of brachial pulsation with palpatory pressure of 55, but without any sound over the brachial and no oscillation of the mercury column, was observed. After the few moments required for the determination of the blood pressure, the patient complained of tingling of the hand in each instance.

DISCUSSION

By title, implication has been made that this interesting set of circumstances may be due to an asymptomatic form of scalenus obstruction, or to other compressions of the arterial trunk as described by Falconer and Weddell and by Wright. The suggestion of Landis that the murmur, being predominant on the left—if indeed this is the same murmur—is due to anatomic variation is well taken. What type of variation? Since none of these cases suffered any symptoms, it was not thought justified to try to explain the problem by surgical attack; only certain rationalizations can be drawn from the substance at hand. It would appear that if the artery were congenitally smaller at its origin, or intrinsically obstructed for any reason, the murmur would be constantly present. Furthermore, a murmur created by intrinsic obstruction would probably be transmitted into the common carotid; this one was not. It was, however, heard posteriorly at the upper border of the scapula, suggesting its transmission through the branches of the third portion of the subclavian artery. It would also seem that fibrous thickening of the dome of the pleura cannot be the commonest cause for the reason that thickening of such an extent should be radiographically demonstrable in a group of young, slender individuals such as these.

Granting that the factor creating the murmur is one of the structures in relation to the artery in its extrathoracic course, none of the presently recognized forms of scalenus compression will explain the progressive obstruction to the artery on elevation of the arm. In fact, it is now generally recognized that among the measures used for the conservative treatment of the clinical syndrome is the maneuver of elevating the arm, usually employed during the patient's sleeping hours. Were it conceivable that the subclavian artery traverses the substance of the scalenus anticus muscle as does an occasional variant of the brachial plexus,[6] then this situation might obtain. Another form of subclavian compression, costoclavicular in operation,[5] was discarded as a possible explanation (one exception) because the murmur was frequently heard with the arm at 45 degrees, the phase in which the clavicle is not in close approximation to the first rib. The authors of this last reference made the observation that their cases showed a relation of position to obliteration of the radial pulse in almost half the cases. The differences between these observations and mine are: first, that the maneuver used was backward and downward bracing of the shoulders, and second, the fact that no murmur was

described, in connection with the changes in pulse, as is described in this communication. It bears out, however, that many persons have changes in circulation, differing from the accepted normal, which are not attended by any symptoms. The human experiments of Harpuda and Stein[8] suggest that no symptoms of ischemia obtain until pressure falls below the usual diastolic pressure for the individual. Since none of these cases demonstrated this degree of obstruction at rest, the absence of symptoms is not surprising.

To demonstrate that cases with cervical ribs were recognized in this series when present, four cases were observed, all bilateral, an incidence of 0.15 per cent. These cases were all asymptomatic, had neither the murmur nor similar changes in blood pressure and pulse.

SUMMARY

1. An attempt has been made to throw some light on the previously described "functional" subclavian arterial murmur.

2. It has been demonstrated that persons with this murmur have definite obstruction of the subclavian artery, usually related to position but not necessarily so, and a characteristic attitude which causes the compression.

3. In the light of limited clinical material, the pro and con of the relation of this murmur to some variant of the scalenus anticus syndrome, costoclavicular compression, or the broader neurovascular syndrome of Wright, are discussed.

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PERIPHERAL NERVE SURGERY

Repair of Nerve Defects

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IN CIVILIAN PRACTICE surgeons are not often called upon to repair peripheral nerve injuries. Such patients as are seen usually have the type of injury which produces a minimal gap between the nerve ends that have to be sutured. Consequently, it is understandable that when a patient with a large nerve defect is presented, the surgeon is apt to be at a loss to answer the questions of whether the defect can be overcome and what methods must be used to accomplish it. During wartime, as happened in the last war, surgeons in large numbers, who had no previous experience, were called upon to do peripheral nerve surgery. In most instances, the nerve defects to be repaired were formidable ones. Unfortunately, these surgeons had no available source of detailed information stating the size defects which could be repaired successfully nor could they find information on the specific procedures to employ in a given instance for overcoming the defect. As a result, errors were frequently made.

At times nerve injuries were classed as irreparable, when a primary suture could have been done. Probably of equal importance is the fact that two-stage procedures were often used in cases that could have been better repaired at a single operation. Nerve grafts were performed (with uniform failure) on some patients who could have had a primary nerve suture with relative ease.

Babcock[1, 2] has listed the approximate gaps which can be overcome and his table was reproduced by Pollock and Davis.[3] Babcock's figures were admittedly estimations, and we found these estimations at considerable variance with our measurements.

It is our purpose in this paper to present accurate information concerning specific defects that can be overcome in all the major peripheral nerves of the body. The extent of the defect will be correlated with the type and length of incision to be employed in overcoming the defect. Secondarily, it is necessary to discuss the principles that must be followed in any operation in which a gap is to be made up between nerve ends. By nerve defect (or gap) we refer to the final defect measured, with the extremity extended, between the proximal and distal stumps after they have been prepared for suture by excision of neuroma from the central end and pseudo-neuroma from the distal segment.

During World War II at the neurosurgical center located at Tilton General Hospital and later at Thomas M. England General Hospital, the writers

collected information on the defects encountered in 625 peripheral nerve sutures performed from April, 1943, to July, 1945. In each instance the defect was measured at operation with the extremity extended and the severed nerve ends as they were found. Then the defect was measured after the two ends of the nerve had been completely mobilized throughout the incision, the ends prepared for suture by excision of neuroma and pseudo-neuroma, and the joints properly positioned. A third measurement was the defect that could have been overcome by utilizing complete mobilization and positioning with the nerve ends sutured under tension, this being the maximum possible defect which could be repaired with any given incision. The figures stated in the accompanying tables are obtained by averaging the defects which have been overcome in many patients and, therefore, it should be pointed out that these figures may vary from one to 1.5 centimeters in certain instances. There are several reasons for this—one is the difference in length of extremities and, more important, is the fact that the relaxation in the proximal and distal ends varies according to the degree of trauma, the severity of the original infection, and the length of time that has elapsed between the injury and the repair.

Nerve defects are overcome by three means: (1) Mobilization of the proximal and distal ends of the trunk. (2) Positioning of the joints of the extremity (usually by flexion). (3) Transposition of the nerve to a new anatomical location. Mobilization and positioning are the methods most generally used to provide the length for bridging a defect. It is only in the ulnar nerve that transposition is commonly done to gain additional length.

It is our belief that considerable judgment must be used by the surgeon in deciding what proportion of mobilization or positioning is to be used in overcoming a defect. By mobilization, we mean the complete dissection of the proximal and distal nerve trunks from the enveloping tissues through the length of the incision. This procedure will necessarily interrupt the small blood vessels that enter the nerve in its course through this part of the extremity. Mobilization can be overdone if one makes an excessively long dissection of the nerve when the proper degree of positioning of the joints would have provided part of the length necessary. An equally serious error is made by employing extensive positioning of the joints at the expense of mobilization. Such is apt to result in excessive suture line tension and, when the extremity is extended, irreparable intraneural damage to the nerve occurs for a considerable distance above and below the point of suture which has a profound effect on the amount of recovery that occurs. Therefore, it is obvious that the proper combination of the two methods must be used in every case. No hard and fast rule can be given for accomplishing this purpose, but the simple realization that the two methods must be employed in their proper relation to each other is a fundamental step toward selecting a satisfactory procedure. In general, mobilization should be considered sufficient when the joint to be used is positioned to approximately 50 per cent of its normal range of motion and the suture can be accomplished without tension.

The figures given in the tables under the heading "Usual Defect Which Can Be Repaired with Ease" represent lengths of defects which have been repaired followed by proven recovery of motor function in the involved nerve. The writers personally followed these cases, and have not been satisfied with questionable signs of recovery. The definite return of motor power has been the only acceptable positive sign of successful nerve suture. The return of some types of sensation cannot be considered an infallible sign of a successful nerve suture with return of function taking place. In the last column in the tables, reference is made to the "Maximum Defect Which Can Be Repaired" with a given incision. This maximum defect has occasionally been made up with proved successful recovery, but more often it represents a hypothetical defect which would have resulted had another few centimeters of nerve been resected in the search to reach normal nerve fibers for approximation. Obviously, this maximum gap will be avoided, if possible, by extending the incision for additional mobilization. At the end of the table for each nerve we have included a statement that represents the greatest possible defect that can be overcome in this nerve by mobilizing the nerve throughout an incision extending the entire length of the extremity and with maximum positioning of the joints. We are quite sure that in the majority of such instances there will be no recovery of function. In a few cases, by employing very careful postoperative extension of the joints, we have had recovery in exceptionally large defects; for example, a 15 cm. defect in an ulnar nerve, a 7.5 cm. defect in a radial nerve, a 12 cm. defect in a median nerve, and a 10.0 cm. defect in a sciatic nerve.

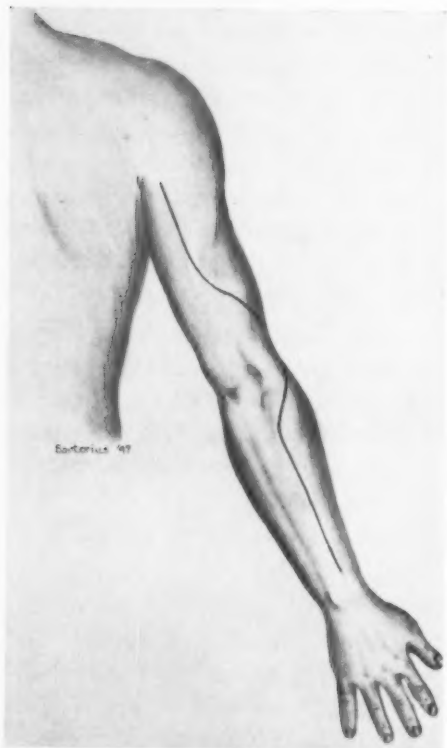

FIG. 1.—Incision for exposure of the radial nerve in the arm and the dorsal interosseous nerve in the forearm. The portion of the incision for exposure of the nerve in the elbow region is shown on Figure 2.

RADIAL NERVE

The incision (Fig. 1) employed in repairing the radial nerve is usually a posterior one which extends from the quadrilateral space laterally around the arm on to the anterior surface between the brachioradialis muscle and biceps brachii tendon to the elbow. Various portions of this incision can be used for small defects, but usually the entire length of the incision will be necessary to

overcome a sizable defect as well as a minimum of 90 degrees of flexion of the elbow joint. The anterior incision is made from the infraclavicular fossa to the upper third of the arm along the course of the neurovascular bundle, and it is used only in those instances when the nerve is obviously damaged in or above the axilla. Occasionally, both exposures will be necessary if the nerve is damaged in the region of the head or neck of the humerus. It is our opinion that transposition of the radial nerve to the medial side of the arm is of little advantage. This will not gain more than one or, at the very most, two centimeters additional length.

It will be noted in the chart that small defects are made up when the lesions are near the elbow joint. This is due to the fact that injury to this region usually involves the bones of the elbow joint and flexion is, therefore, frequently limited so that the final defect which can be made up is consequently less. Another reason is that the injury is often just proximal to the bifurcation of the radial nerve into the superficial and deep branches and this obviates extensive mobilization of the distal segment. The repair of defects in the deep branch of the radial nerve is essentially the same problem as occurs when the main trunk of the nerve is injured in the elbow region.

Injuries to the dorsal interosseous nerve are usually irreparable because more than two centimeters of nerve substance are almost invariably destroyed when there is an associated fracture of the head and neck of the radius, but occasionally when the nerve is cut by a saber or knife wound, it is possible to repair a defect of one or 1.5 centimeters and suture the nerve ends. The current literature indicating repair of defects in excess of two centimeters in this nerve probably refers to lesions of the deep branch of the radial nerve rather than the dorsal interosseous. The dorsal interosseous nerve referred to

TABLE I.—*Radial Nerve*

Information obtained from 79 operations

Site of Lesion	Limits of Incision	Usual Defect Which Can Be Repaired With Ease	Maximum Defect Which Can Be Repaired
Axilla	Supraclavicular region to mid ⅓ of arm	2.3 cm.	2.5 cm.
Arm	Posterior axillary fold to upper ⅓ forearm	6.2 cm.	8.0 cm.
Elbow region	Mid ⅓ of arm to mid ⅓ forearm	3.4 cm.	5.0 cm.
Forearm (dorsal interosseous nerve)	Elbow to distal ⅓ of forearm	1.0 cm.	1.5 cm.
Theoretical maximum defect that can be repaired	Anterior and posterior incisions combined	8.0 cm.	10 cm.

in this paper is that portion of the deep branch of the radial nerve that begins at the inferior border of the supinator muscle after the deep branch has extended around the neck of the radius and extends distally on the dorsal surface of the interosseous membrane to the middle or distal one-third of the forearm. If for any reason a defect in the dorsal interosseous nerve over 2 centimeters is obvious, tendon transfers are done in an effort to overcome the disability due to the paralysis.

MEDIAN NERVE

The incision (Fig. 2) for repair of the median nerve is made from the apex of the axilla along the course of the neurovascular bundle on the medial aspect of the arm to the antecubital fossa and then distally through the middle of the volar surface of the forearm to the wrist. It is only with huge defects that it would ever be necessary to mobilize the nerve throughout the entire length of this theoretical incision. For the average median repair with the injury in the arm, the incision will be from the axilla to the elbow. For an injury in the region of the elbow, the incision will probably extend from mid-arm to mid-forearm. Lesions in the forearm generally require an incision throughout the forearm and possibly into the lower arm. If the lesion is near or at the wrist, the transverse carpal ligament will have to be divided and mobilization of the distal stump carried distally into the thenar region.

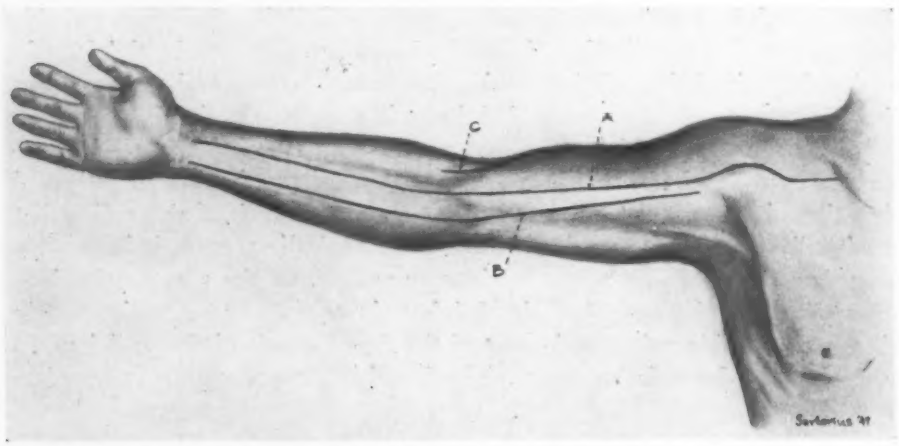

FIG. 2.—A. Incision for exposure of the infraclavicular portion of the brachial plexus the neurovascular bundle in the arm, and the median nerve in the antecubital fossa and forearm. B. Incision for exposure of the ulnar nerve in the arm and forearm. C. Distal end of the incision for exposure of the radial nerve in the arm.

Lesions that occur in the upper part of the forearm frequently necessitate transposing the median nerve superficial to the pronator teres. This is accomplished by separating the muscle from its insertion into the radius and at the same time dissecting the motor branches from the main trunk as far proximally as can be accomplished. This allows a virtual subcutaneous transposition of the nerve and will gain from five to six centimeters additional length. Transposition may be used, of course, with any lesion where additional length is needed. Therefore, it is possible by complete dissection of the median nerve from wrist to axilla and transposition subcutaneously to overcome a huge defect in the neighborhood of 16 to 17 centimeters. This implies that complete positioning is also used by flexing the elbow and wrist. Without transposition, an incision throughout the length of the forearm will afford less than five centimeters of mobilization.

ULNAR NERVE

The incision (Fig. 2) for repair of the ulnar nerve extends from the axilla along the course of the neurovascular bundle passing anterior to the medial epicondyle of the humerus to the upper forearm. That portion of the incision at the elbow should be about one and a half to two centimeters anterior to the medial epicondyle and it must extend distally to the junction of the middle and upper thirds of the forearm if the ulnar nerve is to be transposed anterior to the elbow joint. In the forearm, the incision extends along the ulnar side of the volar surface to the wrist along the lateral border of the flexor carpi ulnaris muscle and ends slightly to the radial side of the pisiform bone. It will be found that in any defect in the arm of more than

TABLE II.—*Median Nerve*

Information obtained from 115 operations

Site of Lesion	Limits of Incision	Usual Defect Which Can Be Repaired With Ease	Maximum Defect Which Can Be Repaired
Arm	Pectoralis major insertion to elbow	6.0 cm.	8.5 cm.
Elbow region	Mid ⅓ arm to mid ⅓ forearm	6.0 cm.	7.5 cm.
Elbow region	Distal ⅓ arm to wrist (nerve transposed)	10.0 cm.	12.0 cm.
Upper forearm	Mid ⅓ arm to mid ⅓ forearm	4.5 cm.	6.0 cm.
Upper forearm	Mid ⅓ arm to mid ⅓ forearm (nerve transposed)	5.5 cm.	8.0 cm.
Mid forearm	Elbow to wrist	4.5 cm.	6.5 cm.
Mid forearm	Elbow to wrist (nerve transposed)	7.0 cm.	9.0 cm.
Distal forearm	Mid ⅓ forearm to wrist	4.0 cm.	5.0 cm.
Wrist	Distal ⅓ forearm to palm	1.8 cm.	2.5 cm.
Theoretical maximum defect that can be repaired	Pectoralis major to wrist (nerve transposed)	12 cm.	17 cm.

two centimeters or in any defect in the forearm of more than four centimeters, it will be necessary to transpose the ulnar nerve anterior to the elbow joint. We prefer making the incision anterior to the elbow joint, but this is a matter of preference, and it can be handled satisfactorily with a posterior incision. We are certain that when the ulnar nerve is transposed it should never be placed beneath the detached flexor group of muscles as is sometimes advocated. Experience has shown beyond any question that this will frequently prevent regeneration, whereas it will progress quite satisfactorily with the nerve in a subcutaneous position external to the flexor group of muscles. It is important to begin the transposition in about the mid-forearm so that the nerve passes through a hiatus in the deep fascia and gradually assumes an anterior position over the flexor group of muscles rather than having it abruptly enter the new position from a point a centimeter or two below the medial epicondyle of the humerus.

OTHER NERVES OF THE UPPER EXTREMITIES

The musculocutaneous nerve is infrequently injured but the lesion occurred 14 times in this series. The usual defect which can be made up with ease in

TABLE III.—*Ulnar Nerve*

Information obtained from 215 operations

Site of Lesion	Limits of Incision	Usual Defect Which Can Be Repaired With Ease	Maximum Defect Which Can Be Repaired
Arm	Axilla to lower ⅓ arm	2 cm.	3 cm.
Arm	Axilla to upper ⅓ forearm (nerve transposed)	6 cm.	10 cm.
Elbow region	Mid ⅓ arm to mid ⅓ forearm (nerve transposed)	5.2 cm.	9 cm.
Upper forearm	Upper ⅓ arm to mid ⅓ forearm (nerve transposed)	4.6 cm.	8 cm.
Mid forearm	Upper ⅓ forearm to wrist (not transposed)	3.2 cm.	5 cm.
Mid forearm	Upper ⅓ forearm to wrist (nerve transposed)	4.7 cm.	7.5 cm.
Distal forearm	Distal ⅓ arm to wrist (not transposed)	3.4 cm.	5.0 cm.
Distal forearm	Distal ⅓ arm to wrist (nerve transposed)	6.5 cm.	10 cm.
Maximum defect which can be repaired	Axilla to wrist (nerve transposed)	12 cm.	16 cm.

the musculocutaneous nerve is four centimeters or less, although it is possible to make up a defect of five centimeters without too great difficulty. The nerve can be exposed throughout its entire length by an incision from the apex of the axilla along the course of the neurovascular bundle to the mid-arm. The defect is then overcome by flexion of the elbow and marked adduction of the arm. Lesions of the axillary nerve are extremely rare unless as part of a brachial plexus injury. In our experience, repair of this nerve is uniformly unsuccessful but a defect of approximately two centimeters can be bridged.

SCIATIC NERVE

The incision (Fig. 3) for repair of the sciatic nerve extends along the middle of the posterior surface of the thigh, beginning at the gluteal fold and extending distally to the middle of the popliteal space. If the lesion is situated above the gluteal fold, it is necessary to detach the gluteus maximus insertion and a different incision is then employed. This is the so-called Stookey operation. If the buttock is not reflected, the incision will probably extend the entire length of the thigh. By reflecting the buttock, it is possible to suture lesions actually within the sciatic notch or at any point distal to it. It is not

TABLE IV.—*Sciatic Nerve*

Information obtained from 53 operations

Site of Lesion	Limits of Incision	Usual Defect Which Can Be Repaired With Ease	Maximum Defect Which Can Be Repaired
Buttock	Buttock to upper ⅓ leg	8.5 cm.	11.0 cm.
Buttock	Buttock to mid ⅓ thigh	6.0 cm.	9.0 cm.
Thigh	Gluteal fold to knee	6.0 cm.	9.0 cm.
Maximum defect which can be repaired	Buttock to upper ⅓ leg	8.5 cm.	11.0 cm.

always possible to repair a very large defect if the lesion is high under the buttock or within the sciatic notch, because it is impossible to mobilize the central stump to any appreciable extent. In fact, if the lesion is more than a centimeter within the sciatic notch, it probably cannot be repaired at all. In addition, in lesions within the sciatic notch, it is impossible to place sutures on the ventral surface of the nerve, and one has to be content with a suture of approximately two-thirds of the dorsal circumference of the nerve. To obtain the necessary length when the lesion is at the notch or under the buttock, it is often necessary to sacrifice one or more of the distal motor branches, usually the branches to the long head of the biceps femoris muscle. This will always give two to three centimeters additional length to aid in overcoming the gap. In spite of the tremendous additional length that flexion of the leg on the thigh will afford, the nerve will sometimes be under such tension that one or two centimeters additional length will be essential, and in order to gain this, hyperextension of the hip is employed. In such instances, it is essential that the patient be put in a spica cast with the hip in the hyperextended position. Maintaining this position during the application of the cast may be quite difficult, but if care is used, one or two centimeters of additional length can always be obtained by this method.

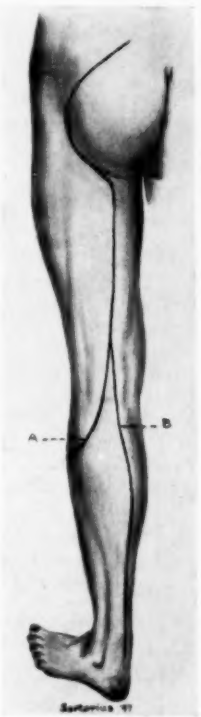

FIG. 3.—Incision for exposure of the sciatic nerve in the buttock and in the thigh. A. Distal end of the incision for exposure of the common peroneal nerve in the popliteal space and upper leg. B. Incision for exposure of the tibial nerve in the popliteal space and in the leg. The distal one-third of this incision is placed midway between the internal malleolus and the Achilles' tendon, more medial than indicated in the illustration.

COMMON PERONEAL NERVE

The incision (Fig. 3) for repair of the common peroneal nerve is identical to that of the sciatic nerve except that in the popliteal space the incision extends laterally, parallel to the biceps femoris tendon and then courses diagonally across the upper leg at the level of the neck of the fibula anteriorly to the interval between the tibia and fibula. We have had no experience with excision of the head and neck of the fibula to gain length for suture, but it seems probable that some additional length could be obtained by this procedure, perhaps two centimeters. It should be noted that repair of the common peroneal nerve in the thigh is no different from repair of the sciatic nerve in the same part of the thigh except that if the tibial portion should be intact, a little less length can be made up in the common peroneal because the intact tibial compromises the dissection somewhat. This results from the anatomic

structure of the sciatic nerve in that the common peroneal and tibial nerves cannot be separated completely from each other throughout the thigh because of intercommunicating fibers connecting the two portions of the nerve. Injuries in the region of the head and neck of the fibula frequently traumatize the common peroneal nerve just at the point where it branches into the superficial and deep portions, and, not infrequently, there is an associated fracture of the bones in this region. Consequently, the repair may resolve itself into a suture of one central branch to two or even more distal branches of the deep component of the nerve. Repair of the deep branch of the common peroneal nerve is technically quite difficult. The dissection is tedious; the nerve is extremely deep between the tibia and fibula on the interosseous membrane beneath the tibialis anterior and the extensor communis digitorum

TABLE V.—*Common Peroneal Nerve*

Information obtained from 97 operations

Site of Lesion	Limits of Incision	Usual Defect Which Can Be Repaired With Ease	Maximum Defect Which Can Be Repaired
(Proximal to the distal ⅓ of thigh defects are the same as the sciatic nerve)			
Distal ⅓ thigh and popliteal region to neck of fibula	Gluteal fold to neck of fibula	6.4 cm.	8.1 cm.
Leg (distal to neck of fibula) Superficial branch	Popliteal space to mid ⅓ of leg	1.3 cm.	2.5 cm.
Leg (distal to neck of fibula). Deep branch	Popliteal space to mid ⅓ of leg	1.0 cm.	1.5 cm.
Maximum defect which can be repaired	Buttock to upper ⅓ leg	8.5 cm.	11.0 cm.

muscles; and suture is difficult even if the nerve ends are near approximation because the nerve is small. If there is a defect of any significant size the nerve cannot be repaired. Rarely is it possible to bridge a defect as great as 1.5 centimeters. The dissection of the superficial peroneal branch after it extends distal to the neck of the fibula is not difficult, but again only a small defect can be repaired—at most 2.5 centimeters.

TIBIAL NERVE

Incision (Fig. 3) for repair of the tibial nerve in the popliteal space or in the lower thigh is the same as for repair of the sciatic nerve. Less defect can be made up in repair of this nerve in the thigh than in the sciatic nerve because it is necessary to separate extensively the tibial from the common peroneal component. This is not advisable if it can be avoided because of the intercommunicating fibers that will be damaged, and disrupting the epineurium on half the circumference of the nerve adds to the technical difficulty in accurately suturing the ends. In the popliteal space less defect can be made up in the tibial nerve than in the common peroneal nerve because its course is more direct and it is more deeply situated so that flexion will not provide the additional length that will be gained in the longer and more superficial common

peroneal nerve. To repair the tibial nerve in the leg, an incision is made beginning at the ankle midway between the medial malleolus and the Achilles' tendon extending proximally in a gradual curve to the midline of the leg over the belly of the calf muscles and gradually back to the midline of the popliteal space. In the leg if the defect is greater than five centimeters it is necessary to carry out a very extensive dissection which involves detachment of the soleus and gastrocnemius muscles from the posterior surface of the tibia up to the popliteal space, and retracting the entire muscle mass laterally. This exposes the nerve which is deeply situated in the center of the calf. By this procedure a very large defect can be repaired if the incision is extended from the

TABLE VI.—*Tibial Nerve*

Information obtained from 52 operations

Site of Lesion	Limits of Incision	Usual Defect Which Can Be Repaired With Ease	Maximum Defect Which Can Be Repaired
(Proximal to the distal ⅓ of thigh defects are the same as the sciatic nerve)			
Lower thigh	Gluteal fold to mid ⅓ leg	5.2 cm.	6 cm.
Popliteal region	Gluteal fold to mid ⅓ leg	4.5 cm.	9 cm.
Leg	Upper ⅓ leg to ankle	3.0 cm.	5.0 cm.
Leg	Distal ⅓ thigh to ankle	8.0 cm.	11.0 cm.
Malleolus	Mid ⅓ leg to malleolus	1.5 cm.	2.5 cm.
Maximum defect which can be repaired	Buttock to upper ⅓ leg	8.5 cm.	11.0 cm.
	Mid ⅓ thigh to ankle	9.0 cm.	12.0 cm.

malleolus through the popliteal space into the lower thigh; as much as 11 or perhaps even 12 centimeters can be made up by this maneuver. It is rare, however, that a defect in excess of six or seven centimeters is encountered in this nerve. In lesions of the medial and lateral plantar nerves near or below the internal malleolus, only small defects, one or two centimeters in size, can be repaired. Flexion of the knee joint in the extensive dissection supplies considerable additional length not gained by mobilization alone, while plantar flexion and inversion of the foot accomplish very little in bridging defects over one centimeter long.

SUMMARY

1. Accurate data on the defects that can be bridged in all the major peripheral nerves of the body are presented.

2. The fundamental technical methods employed in overcoming nerve defects are discussed.

3. Practical considerations in repair of a defect of each major nerve are suggested.

4. Summaries are presented for each nerve in accompanying tables.

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MYXOMA, THE TUMOR OF PRIMITIVE MESENCHYME*

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TUMORS OF A MUCOID or myxoid habit resembling mesenchyme are not too uncommon. In a recent study of soft part sarcomas they stood in third place among sixteen named varieties, being surpassed only by fibrosarcoma and liposarcoma (Stout, 1947). Yet the literature contains very little accurate information about them as an entire group. There are several reasons for this. In the first place, there are a number of tumor forms which sometimes are composed in part of myxoid tissue such as liposarcoma, fibrosarcoma, chondrosarcoma, and mesenchymoma. Such tumors sometimes have "myxo-" included as part of the name and there has been a tendency to consider them as variants of the myxoma. This is certainly an error, for clinically they behave like tumors composed of the dominant tissue and should be designated by that name. Further, there has been a tendency on the part of some to use both the terms myxosarcoma and myxoma. This also is probably unwise, for myxomas do not metastasize and there is no way to anticipate differences in their growth energy from their histopathology. Still another difficulty arises because of the resemblance of ganglions of the tendon-sheaths and skin to myxomas. Finally, there are the peculiar primary myxoid tumors of the heart, about which so many papers have been published denying and affirming their neoplastic nature, their degree of malignancy and their relationship to primary heart muscle tumors. It seems probable that among them there are examples of undifferentiated mesenchymal proliferations both granulomatous and neoplastic. Of the latter some are undifferentiated myxomas, some are rhabdomyomas, and some are mixed (Batchelor and Maun, Mahaim, Ravid and Sachs, Anderson and Dmytryk). It must also be pointed out that myxomas of the heart are the only tumors given that name from which metastases have been reported (Fenster). This is so exceptional as to warrant the suspicion that such metastasizing tumors are probably not true myxomas but sarcomas of some other type masquerading as myxoma. For these reasons it is quite difficult to learn accurately from the literature the exact distribution and biological course of this group of tumors.

It seems necessary first to define what shall be understood by the term "myxoma." For the writer, it is a true neoplasm composed of stellate cells set in a loose mucoid stroma through which course very delicate reticulin fibers in various directions. In other words, it closely resembles primitive mesenchyme. This resemblance has been noted by Greco, by Harris, by Hogenauer, by Saturski and others. The only variation from this is the occasional

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formation of denser areas due to a thickening of the delicate connective tissue fibers and a lessening of the mucoid material. In such areas, because of this increase in density, some of the cells may become spindle shaped, probably as a result of pressure molding, but these areas should not be extensive. One must allow this much variation, since any tumor may undergo some degree of fibrosis. There must be no chondroblasts, lipoblasts, rhabdomyoblasts or any other recognizable differentiated elements. Unlike the progressing ganglions which have a multicentric origin and tend rapidly to form cysts containing hyaluronic acid, the myxoma has a unicentric origin, grows progressively by infiltration and/or expansion and rarely reaches a great size. It is probable that the mucoid material of the myxoma is hyaluronic acid and not mucus. The fact that when hyaluronidase was added to the thick material obtained from the tumor in Case 1 this material was partly fluidified, would suggest this. According to Meyer there is a relatively large amount of hyaluronic acid in the primitive mesenchyme, which also suggests that the myxoma is a neoplastic reproduction of primitive mesenchyme. Perhaps most important of all, the myxoma does not metastasize and, if it kills, it is because of damage to vital structures produced by infiltrative or expansile growth causing pressure or erosion. As examples of this there may be cited the case of Hogenauer (1933) in which death from asphyxia was caused in a 40-year-old woman by a myxoma of the neck that surrounded the esophagus and completely blocked the trachea by compression, and Rosenberg's (1936) 46-year-old patient with myxoma of the prostate which caused death by filling the pelvis and compressing its structures.

Using this definition, the writer has been able to recognize 49 cases of myxoma recorded in the Laboratory of Surgical Pathology of Columbia University, and after a not too exhaustive search of available literature has found 95 more, exclusive of the heart. More than 100 cases of myxoma involving the heart have been reported.

The anatomic distribution of these cases is shown in Table I.

An examination of this table shows that most of these tumors are found in the heart, the skin, subcutaneous and aponeurotic tissues in certain bones, and the genitourinary system. Myxomas in other situations are rare. The soft part tumors are widely distributed, but many of the bone cases are found in the jaws. In addition to the bones mentioned in the chart, cases are reported in the fibula (Leriche), tibia (Lehmann), ulna (Garavano and Schwjowicz), skull (Brackmann; Inclán and Inclán), jaws (Thiargo Marques; Tholen, Milhon and Parkhill), metatarsal (Copello), periosteum of metatarsal (Marziani) and phalanx of toe (Danielewski and Komza). Other bone cases were described by Bloodgood. In the genitourinary system, the greatest number are found in the urinary bladder, spermatic cord and vulva.

The sex of 46 of our cases is recorded: 25 were female and 21 male. Six patients were Negroes. The age at onset of symptoms is recorded in Table II. From it one can only conclude that the myxoma is a tumor which may make its appearance at any time from birth to old age.

Clinically the tumors in the soft parts which can be examined do not have any very striking characteristics, largely perhaps because most of them are so deep that their myxoid qualities are masked, or, if they are in bone, completely hidden. The largest tumor in the Columbia University group

TABLE I.—*Anatomic Distribution of 140+ Myxomas*

	Total	Personal	Lit.
Subcutaneous and aponeurotic	32	25*	7
Bone	26	10†	16
Genito-urinary	23	2	21
Skin	22	4	18
Retroperitoneal	5	2	3
Intestine	5	1	4
Nares and sinuses	5	0	5
Muscle	4	1	3
Joint	4	1	3
Pharynx and tonsil	3	0	3
Breast	3	0	3
Orbit and eyelids	4	1	3
Intracranial	1	1	0
Spleen (Tománek)	1	0	1
Appendix (Laird and Nolan)	1	0	1
Liver (Zuidema and Seldam)	1	0	1
Parotid gland (Valensin)	1	0	1
Carotid body (Bertola)	1	0	1
Ear (auricle) (Hand and O'Connor)	1	0	1
Heart	many	1	many
	143+	49	95+

* Lower ext. 7, upper ext. 6, head 5, back 5, neck 1, inguinal 1.
† Mandible 5, maxilla 3, clavicle 1, metatarsal 1.

measured 30 x 10 cm. and was in the leg of a 68-year-old male. It had been excised when smaller, after 19 months of growth. Recurrence appeared after one month, and in four more it had attained the size recorded. The patient refused amputation and could not be traced after leaving the clinic. The largest authentic myxoma recorded was reported by Jonas (1937). After

TABLE II.—*Age at Onset of Symptoms of 99 Cases of Myxoma*

0–9	10–19	20–29	30–39	40–49	50–59	60–69	70
17	11	11	10	25	11	13	1

three years of abdominal enlargement a tumor measuring 32 x 26 x 24 cm. and weighing 5426 gm. was removed from the retroperitoneal region of a 36-year-old woman. It originated seemingly from the parametria and extended upward to both sides of the diaphragm. It was gelatinous with cystic areas, and histologically was a vascular myxoma. No follow-up was reported. A tumor weighing 82 kilos attached to the labium majus, extending up between vagina and rectum and measuring 62 x 63 cm. with a circumference of 125 cm. was described by Leischner (1930) but this was probably a liposarcoma.

The duration of symptoms (*i. e.* tumor) before treatment has varied widely from two weeks to 37 years and averaged 4 years. Like many other tumor varieties it is apparent that a myxoma tends to grow very slowly or remain stationary for long periods of time and then may suddenly enlarge rapidly. There is no rule about the relationship of these phases one to the other; rapid growth may come at the beginning or the end of a quiescent period, or may be both preceded and followed by inactivity.

It is of interest to find that both Krogius and Bolognesi have noted the concurrence of myxomas in the soft parts and fibrous lesions in the bones. When myxomas grow in bone, they develop in the marrow, expand the cortex, destroy bone by aseptic pressure necrosis and produce a deformity of the bone which cannot be distinguished roentgenologically from osteitis fibrosa, giant cell tumor or fibrous dysplasia, and, in the case of the jaws, from adamantinoma or paradental epithelial cyst (Fig. 5). If, as in Case 5, the tumor springs from the periosteum, there may be no bony deformity (Fig. 7). When myxomas grow in or close to the skin, they have a semitranslucent pallid aspect which simulates the appearance of some lymphosarcomas and ganglions of the skin and of an occasional liposarcoma, if invasion brings it close to the epidermis. In this situation the myxoma may be soft and suggest fluctuation (Fuhs, Gross, Jacox and Freedman, Kusnetz, Maynard, Sanchez Carvisa and Bejarano). While many of them are found at the finger tips (Case 2), they have been reported from other parts of the skin surface in both single and multiple form. Almost all of them are relatively small. In the intestine most of them have been pedunculated growths projecting into the lumen and causing intussusception (Case 6, Brachetto-Brian and Latienda, Sullivan and Corcoran, Du Bourguet *et al.* Perry and Peters). Most of the genitourinary myxomas have developed in infants from the bladder (Bongiorno, Grynfeltt, Harris, Lazarus and Rosenthal, Meade, and Weiss and Meyer) but Saturski's patient was 33 years old. The trigone was the usual site of origin. Hematuria and obstructive symptoms occurred and usually, although not invariably, growth progressed continuously to a fatal outcome in spite of the most radical attempts at removal. Myxomas have been found in the spermatic cord (Baiocci, Collins and Berdez, Tsuchiya and Shindo), the scrotum (Grimaldi and Bernardi, Loubat and Dareys, Menville), the round ligament (Peltier de Queiroz), the vulva (Abdanski and Landsberg, Menini) and the ovary (Kikuti and Minakawa). Orbital and eyelid myxoma cases have been reported by Lamb, Quintana and by Town, mammary gland myxomas by Marano, Posgay and by Sammartino and there are several reports of myxomas in the upper respiratory passages. The larynx cases are all simply myxomatous polyps and the nasopharyngeal cases either polyps or the fibroangiomas of adolescents. Fuste and Mena Serra's tumor of the maxillary sinus may be a true myxoma and the same may be said of Richter's and of Shiroto's tumors of the antrum.

The treatment of most of these patients has been by surgical excision. Radiation therapy, whether by radium or roentgen-ray, has generally been

either entirely or partly unsuccessful as, for example, in Tománek's patient who had a myxoma of the spleen reduced to one-half its original volume with external treatment by radium, and in Puente Duany and Paultre's tumor of the thigh treated by roentgen-ray. However, Jacox and Freedman, by using caustic doses of roentgen-ray for the small finger lesions, reported that they effected cures. Sometimes the operative removal has been adequate but too frequently it has been inadequate, as evidenced by many examples of recurrence. The following examples are illustrative: Dixon and Vadheim removed a grapefruit sized myxoma from the region of the kidney. In spite of postoperative roentgen-ray treatment, a recurrent mass adherent to the hepatic flexure necessitated a partial colectomy for its removal 7½ years later. Hand and O'Connor's patient had several recurrences involving the external ear, which finally resulted in its loss after 4½ years. Trabucco described a myxoma of the lateral neck region which recurred eight times in 35 years. One of the patients in the group here reported, a man 65 years old, had eight operations for the excision of recurring myxoma of the lateral neck region lasting over a period of 36 years. The results obtained can only be indicated because the follow-up data at hand are inadequate. They are shown in Table III.

TABLE III.—*Results of Treatment of 27 Followed Cases of Myxoma Recorded in the Laboratory of Surgical Pathology of Columbia University*

Alive without tumor	—over five years	5*
	under five years	9
Alive with tumor	—over five years	3**
	under five years	7
Died	—following operation	1
	because of tumor	1
	of intercurrent disease with tumor persisting	1
Total		27

* Mandible 5 and 23 years, maxilla 5½ years; arm 6½ years; thumb 17 years.
** Mandible 9 years, leg 16 years, lateral neck 36 years.

An examination of Table III shows the inadequacy of many of the operations for removal of myxomas. In some instances this may not have fatal results because these tumors do not metastasize and the recurrences in non-essential parts of the body may only be a source of annoyance and discomfort. But sometimes the recurrences may cause death, for instance, if the tumor is in the bladder or retroperitoneal region or at the base of the skull where it is impossible to remove all of its extensions and where persistence of growth can interfere with vital functions. The writer has long been of the opinion that the only proper way to deal with the various tumors derived from mesenchyme is to biopsy them before undertaking treatment. When study of the paraffin section reveals the nature of the growth, a knowledge of the possible behavior of the particular variety of tumor demonstrated will serve as a guide for planning treatment. With most myxomas it is usually necessary to remove a generous amount of apparently uninvolved surrounding tissue to effect eradication.

ILLUSTRATIVE CASES

Case 1.—S. P. 90331. L. G., an 18-year-old colored girl from British Guiana first noticed a lump about the size of a marble in her right arm, 3 years before admission. Her mother said it had been present since birth. Two months ago it caused some mild discomfort. A deeply seated mass which made a visible lump was found on examination in the region of the insertion of the right pectoralis major muscle into the humerus. It was firm, not tender and was movable (Fig. 1). The Kline test was negative. The bone was not involved.

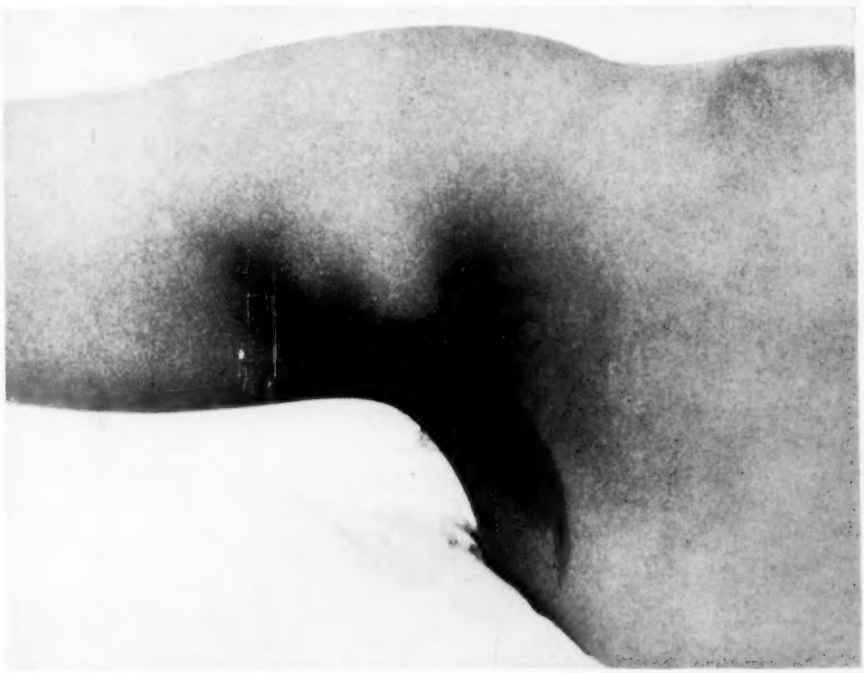

FIG. 1.—Case 1: Myxoma of the arm.

At operation 7/29/44, the tumor appeared encapsulated and lay in the tendon of the pectoralis major muscle near its insertion into the humerus and in the adjacent fascia. The periosteum was not involved. The tumor was first carefully exposed and a biopsy was taken. It was mucoid in character and a quick frozen section was diagnosed myxoma. A second incision was then made through which the first wound, the tumor and the tissues surrounding it were removed in one block without again exposing the tumor. This necessitated removal of a portion of the tendon of the pectoralis major muscle and some of the surrounding fascia. There was no recurrence or interference with function when the patient was last seen 3/25/47, 2 years and 8 months after operation.

Gross examination of the tumor showed that it was apparently encapsulated, measured 4 cm. in diameter and on section its consistency was somewhat fibrous, sticky with mucoid and had the color of weak lemonade. The microscopic picture shows a loose-textured tissue of tangled reticulin fibers, somewhat thicker than in other myxomas, separated by clear spaces containing mucoid and set at intervals with stellate cells. The capsule was not invaded by the tumor (Fig. 2).

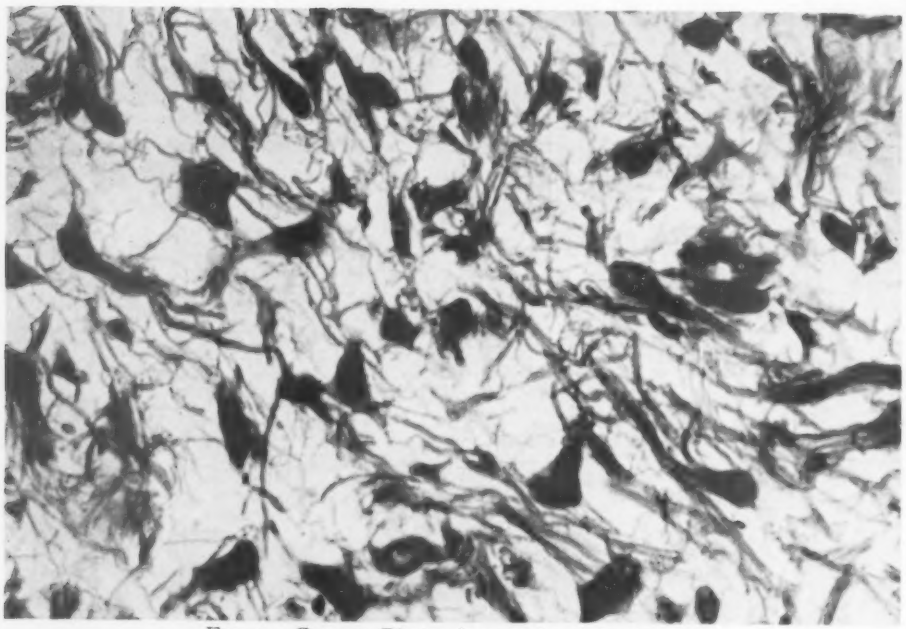

FIG. 2.—Case 1: Photomicrograph of the tumor.

FIG. 3.—Case 2: Myxoma of the finger tip.

Case 2.—S. P. 94723. H. S., a colored male shipping clerk born in Curaçoa, D. W. I., 31 years old, came to the Vanderbilt Clinic complaining that 3 years before he had burned the tip of his left index finger and subsequently bit and scratched the area. Some time after this a soft swelling appeared which slowly increased in size. Examination showed a tense fluctuant mass on the lateral volar aspect of the tip of the left index finger. It measured 1 cm. in diameter and was called a skin ganglion (Fig. 3). August 20, 1945, it was excised intact and the wound was grafted. Nine days later the graft had taken and all sutures were removed. He did not return to the Clinic for further observation.

The specimen removed measured 1.9 x 1.2 x 1 cm. and two-thirds of its surface was covered by epidermis. On section it showed a glistening translucent mucoid surface. Microscopic examination showed an encapsulated growth lying in the skin, elevating the epidermis and composed of a very loose-textured tumor with fine tangled reticulin fibers, abundant mucoid material and widely spaced stellate cells (Fig. 4).

Case 3.—S. P. 26923. M. P., a 13-year-old school girl of Italian parentage had had trouble with the lower left first molar tooth for some years. Five weeks before admission she noted swelling of face in this region. One week before admission the loosened molar tooth was extracted. Examination on admission showed that the alveolar process of the left mandible and the bone beneath it were swollen from the second bicuspid

through the second molar regions. Roentgen-ray showed an area of rarefaction and expansion of the mandible in this region with displacement of tooth roots. The bony walls of the cavity were thickened by very delicate ridges. The inferior cortex of the body of the mandible appeared very thin (Fig. 5).

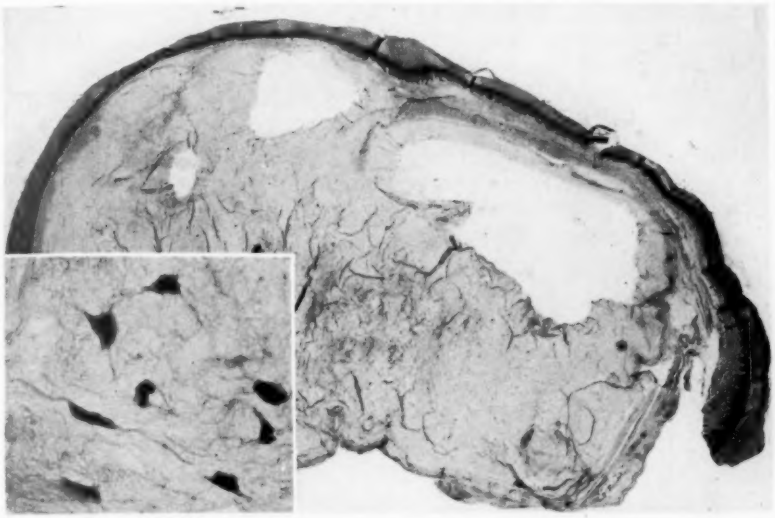

FIG. 4.—Case 2: Photomicrograph of the entire tumor. The inset shows details of cells and stroma.

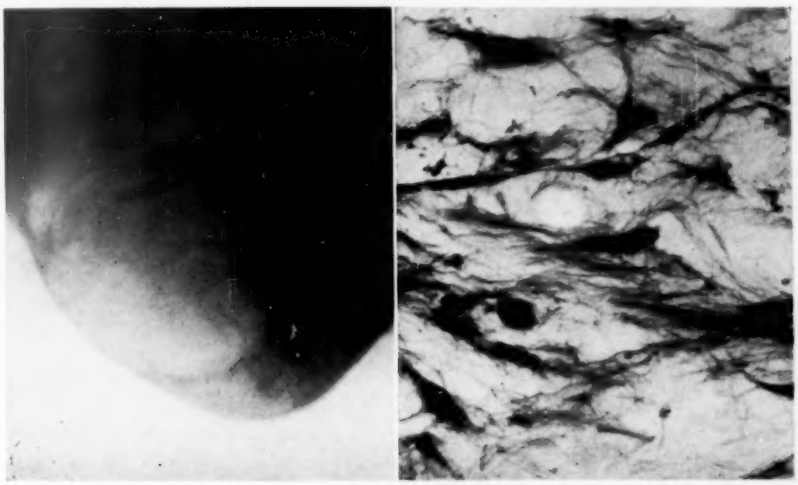

FIG. 5.—Case 3: Roentgenogram of myxoma of mandible and photomicrograph showing detail of histopathology.

At operation 12/14/21 the cavity was entered through the alveolus and a soft, gelatinous, semitranslucent, pale grayish tumor was encountered and excised piecemeal, together with some bony trabeculae which partly divided the cavity. Five weeks after operation, radium in tubes was placed in the open cavity, and a dose of 400 mgm. hours was given. There was no recurrence when the patient was seen 23 years after operation.

Microscopic examination showed a tumor composed of a stroma of tangled delicate reticulin fibers with mucoid material in the intervening spaces and scattered stellate cells (Fig. 5).

Case 4.—L. L., a 26-year-old married American Negress was admitted to the Presbyterian Hospital 6/11/41. Nine months before she noted a small lump in the anterior aspect of the right mandible which gradually increased in size, causing a sense of fullness and occasional shooting pains.

On examination an egg-shaped mass was observed in the mandible extending from the lower left second premolar to the lower right premolar. It pushed the teeth backward

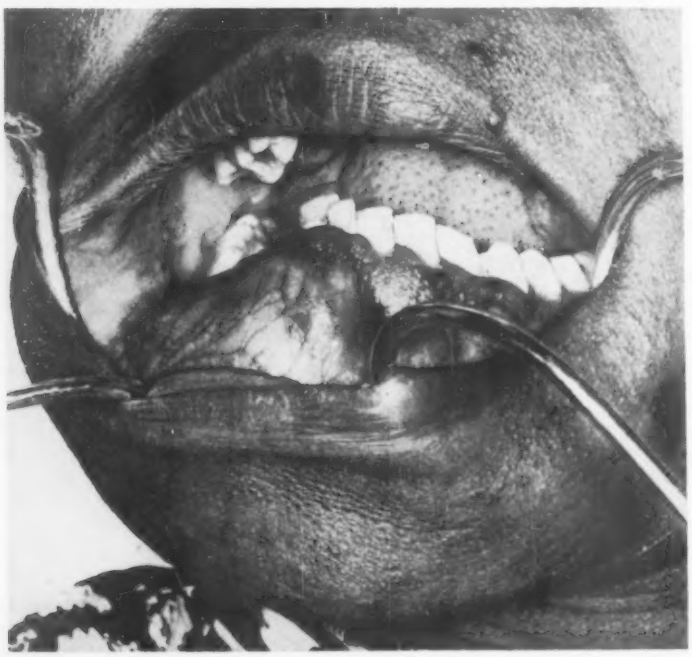

FIG. 6.—Case 4: Photograph of intraoral appearance of myxoma of mandible.

and the lip forward (Fig. 6). June 12, 1941, the mandible was resected between these two points.

On gross examination the cortical bone was exceedingly thin anteriorly and the teeth loosened. The tumor inside the bone was homogeneous, reddish gray and translucent. It occupied almost all of the bone resected and came very close to the lines of resection. October 3, 1946, five years and 4 months later there was no recurrence.

Microscopically the tumor was a characteristic myxoma composed of stellate cells set in a loose mucoid stroma with delicate reticulin fibers in it. Occasional bony trabeculae were noted throughout the tumor.

Case 5.—S. P. 85510. L. L., British West Indies Negro 50 years old. For the past 15 years the patient had had a growth attached to the left clavicle which appeared without any known cause and remained stationary until 2 years ago when it increased slowly in size but was symptomless.

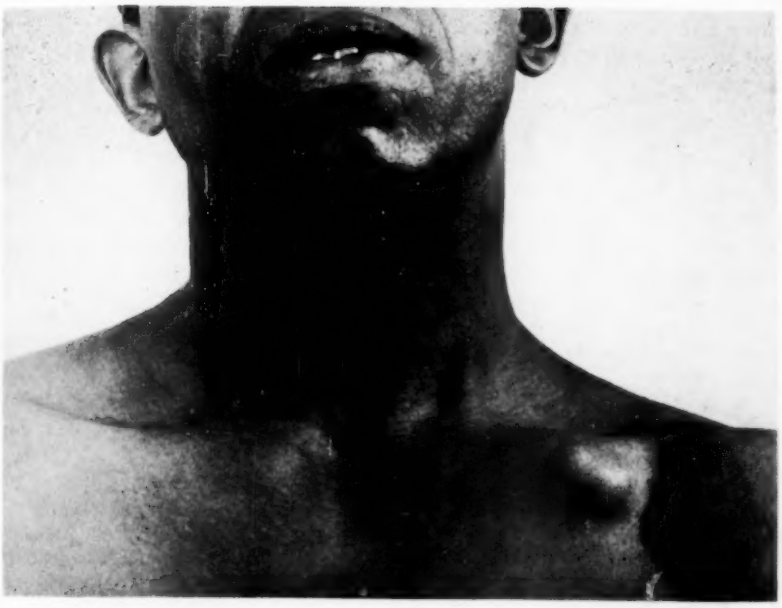

FIG. 7.—Case 5: Myxoma of periosteum of the clavicle.

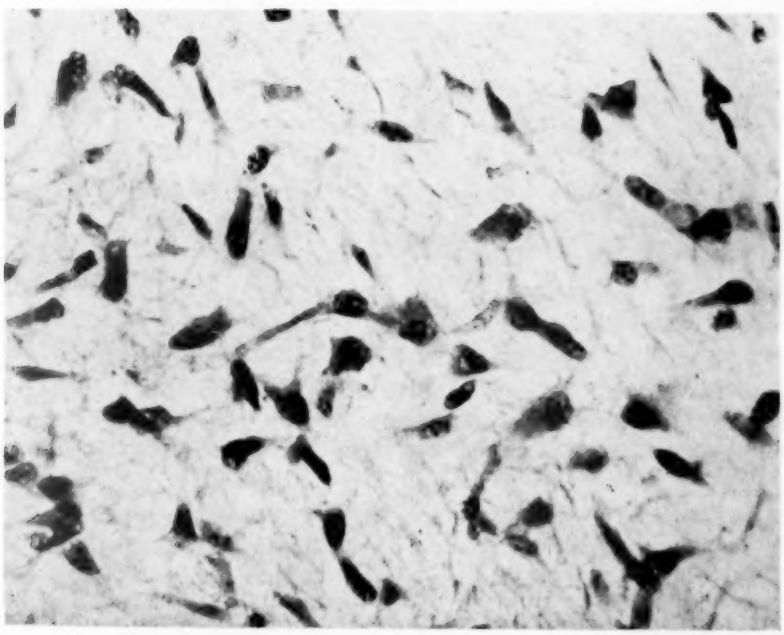

FIG. 8.—Case 5: Photomicrograph showing histopathology of tumor.

On examination there was found an elastic lobulated tumor attached to the middle and inner thirds of the left clavicle. It measured 8 x 5 x 2 cm. Roentgen ray showed no bony lesion of the clavicle (Fig. 7).

Operation 4/26/43. The tumor was ovoid, lobulated and had a gelatinous spongy consistency. It was apparently encapsulated and firmly fixed to the periosteum of the clavicle, which was excised with the tumor together with some of the cortex which appeared unaffected. Fifty months after operation there as no evidence of recurrence.

The nodular mass measured 58 x 33 x 32 mm. and had a small fragment of the cortex of the clavicle attached to it. It appeared semitranslucent, and when cut a clear colorless stringy mucoid material escaped. Some of this was investigated by Dr. Karl Meyer who added to it a little hyaluronidase. This partly liquefied the thick material which suggests but does not prove that it was hyaluronic acid.

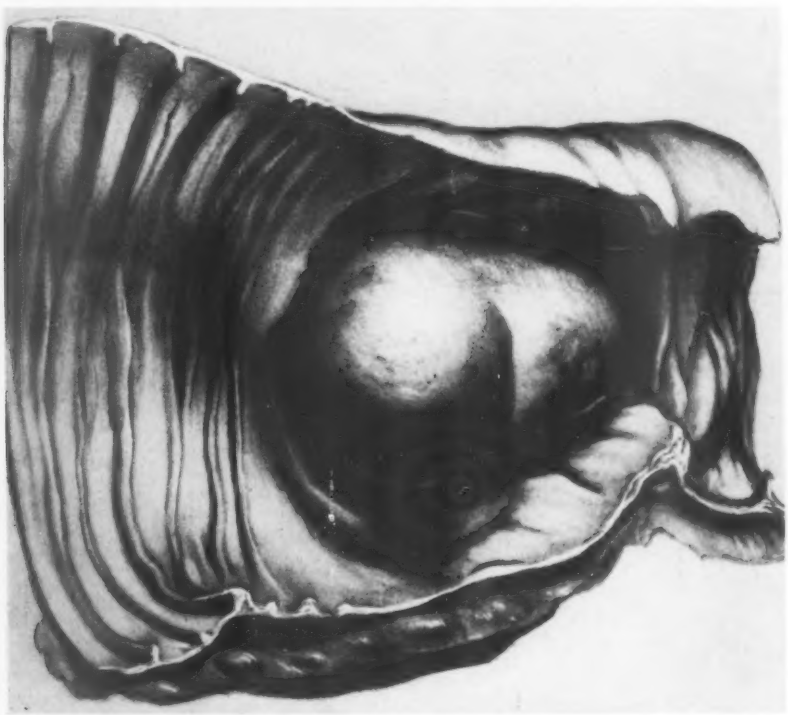

FIG. 9.—Case 6: Myxoma of the ileum.

Microscopic examination showed a very loose textured tumor composed of stellate cells set at wide intervals with mucoid material and a very fine meshwork of reticulin fibers. No lipoblasts were seen. The bone was not invaded (Fig. 8). The tumor was explanted *in vitro* by Dr. Margaret R. Murray. Most of the fragments did not grow at all. Three formed a few stellate cells resembling those found in the tumor.

Case 6.—S. P. 22376 (5394). H. S. A married American housewife 68 years old. For 6 weeks before admission she had suffered from intermittent pains in the umbilical region with vomiting, constipation and distention. Physical examination on admission was unrewarding except for distention. A flat plate of the abdomen showed obstruction in the small intestine. At operation 2/15/19 an intussusception was found in the ileum due to the presence of a pedunculated tumor projecting into the lumen. After easy

reduction 12 cm. of the ileum was resected with end-to-end anastomosis. The patient made a good recovery and was symptom free one month later.

Examination of the specimen showed a pedunculated 4.5 x 3.5 cm. smooth tumor which was moderately firm and apparently sprang from the submucosa (Fig. 9). Sections showed an ulcerated surface covered with granulation and scar tissue which covered the tumor. The latter was composed of stellate cells set in the usual loose textured stroma containing mucoid material and exceedingly delicate reticulin fibers. The tumor appeared well vascularized (Fig. 10).

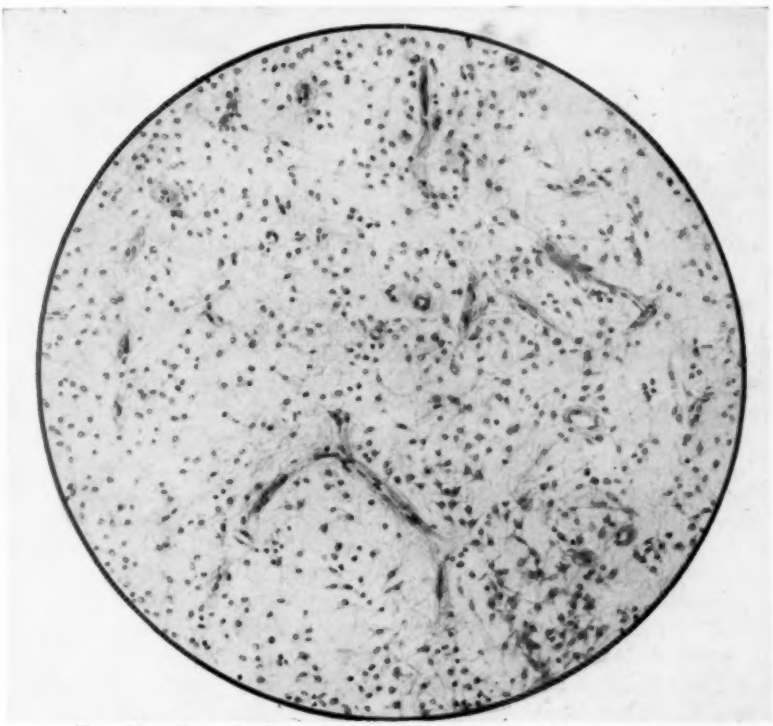

FIG. 10.—Case 6: Drawing showing histopathology of tumor.

SUMMARY

The myxoma is a tumor of mesenchymal origin which reproduces with some degree of accuracy the appearance of primitive mesenchyme. It is made up of a loose textured slimy tissue composed of stellate cells set in a stroma of exceedingly delicate reticulin fibers and a mucoid substance which is probably hyaluronic acid. Rarely portions of it may become denser and resemble fibrosarcoma but no other metaplastic changes occur. The tumor generally infiltrates surrounding tissues to some degree. It is found equally in both sexes at all ages and in many tissues but most frequently in the heart, the skin and soft parts, the bones and the genitourinary system. In the skin the fingers are most frequently involved; a majority of the bone cases are found in the jaws and most of the genitourinary cases develop in the bladder.

The tumors grow at different rates of speed, but usually there are long periods of inactivity which may precede or follow shorter periods of rapid growth. Many never attain a very large size but a tumor weighing over 5 kilos has been described. Metastases have never been reported except from myxoma of the heart and it is possible that this was not a simple myxoma. In any event, their possibility does not have to be considered in treatment. Since the tumors infiltrate, close excision has frequently been followed by recurrence. If this takes place in some region where vital structures can be affected, such as the bladder and retroperitoneal region, a fatal outcome may be the result. The importance of biopsy before treatment in all tumors of mesodermal origin is stressed.

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RAYNAUD'S PHENOMENON AND ATYPICAL CAUSALGIA; THE ROLE OF SYMPATHECTOMY

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It is well to recognize that the employment of sympathectomy has been somewhat empiric, since Alexander,[1] in 1899, first performed a cervical sympathectomy for epilepsy. It is only by the gradual accumulation of experience that the procedure has begun to rest on a firm foundation. Realizing that there is an obvious controversy as to its efficacy in many conditions, such as Buerger's disease and hypertension, it is desired to discuss only its use in cases where there is a marked vasospastic element present, without organic obliteration of vascular channels. This report is concerned with the role of sympathectomy in Raynaud's phenomenon and atypical causalgic states. In these, cold sensitivity is a major manifestation.

Since vasoconstriction is one function of the autonomic nervous system, mediated through the sympathetic fibers, one method of study of vasospasm is based upon the interruption of these pathways by various means. One suspects the presence of vasospasm upon the appearance of the following signs and symptoms:

1. Hyperhidrosis
2. Coolness
3. Cyanosis
4. Cold sensitivity
5. Color changes

Although it is obvious that the mechanism of peripheral vasoconstriction can be humoral as well as neurogenic, it is felt that the latter mechanism is more important. It has, therefore, been considered advisable to test the degree of neurogenic vasospasm by a direct objective method—namely, block of the regional sympathetic ganglia with procaine. For the upper extremity, block of the stellate ganglion by the anterior route with 10 cc. of ½ per cent procaine is done, as this method is easily performed and taught, and the objective manifestations of Horner's syndrome[3] are unequivocal as to the success of the block. A comparison of the method of posterior block in the region of T2 and T3 with this method presents nothing of importance to recommend the former over the latter; in addition, it is much more difficult to perform, and as hazardous because of the danger of the occurrence of pneumothorax. In the lower extremity, I use a single injection of 30 cc. of ½ per cent procaine with a 22 or 20 gauge needle 7 inches long, at a 40° angle to the sagittal plane, in the region of L2 or L3. This method is almost uniformly successful, and not nearly so painful as the three or four needle technic of paravertebral block. Skin temperature determinations, as well as clinical observations, are made under standard conditions of temperature and humidity routinely before and after sympathetic ganglion block.

Although surface temperatures do not accurately reflect the vascular status of the deeper structures,[4] it should be recognized that this distinction is not nearly so important in vasospastic conditions as in obliterative organic diseases, such as Buerger's disease and arteriosclerosis. This has proved to be a helpful objective determination, when correlated with clinical observations of the extremity made before and after block to note improvement in color; and, equally important, amelioration of the major complaint of the patient. Oscillometric determinations have proved no more advantageous than clinical observation of the peripheral pulses in the consideration of the status of the peripheral vascular condition, particularly in primary vasospastic disorders. This has been the experience of others.[5] Other methods of undoubted value in diagnosis and evaluation of peripheral vascular disease are plethysmography and capillary microscopy.

The following types of cases representing various degrees of primary vasospasm have been seen and treated:

	Total No. Seen	No. Operated On	Preganglionic Sympathectomies
Raynaud's Phenomenon	20	5 (25%)	9 cervicodorsal
Atypical Causalgia	6	6	(1 cervicodorsal (5 lumbar
Cold Sensitivity and pain after ligation of main vascular channels (femoral)	2	2	2 lumbar

In all of these cases, the primary condition was one of vasospasm rather than organic obliteration of the major vascular channels and their tributaries. Exception may be taken to this in the third group, where ligation of the main vessels, the femoral artery and vein, had been done; but, in effect, there was no evidence of organic obliteration of the vascular tree distal to the ligation, as is seen in obliterative vascular disease.

In considering the cases amenable to sympathectomy, one is impressed by a common feature which seems predominant, and that is sensitivity to even *moderate cold,* with marked discomfort and cyanosis. In a sampling of a diverse group of 31 cervicodorsal and lumbar sympathectomies done, all but six (80 per cent) presented this as a primary manifestation. Most presented this as an initial complaint. In practically every instance, the objective manifestations became prominent on exposure to cold. The cold did not have to be severe. This was particularly true of the patients with Raynaud's disease, in which the typical symmetrical triphasic color changes could be best produced in a cool environment rather than by immersion of their hands in ice water.

A total of 20 cases of rather severe manifestations of Raynaud's syndrome were originally seen, and from these, five (25 per cent) were selected for sympathectomy. All these cases occurred in men. It is well to emphasize that, although the incidence ratio of women to men is 5:1, and a diagnosis

of Raynaud's disease in men should be viewed with suspicion, the disease is by no means limited to women. Evidence to this effect is given by the report of Hines and Christensen[6] in which 198 (23 per cent) of 847 cases seen were men. It is an error to teach that the disease rarely occurs in men. The cases concerned in this report, conform to the criteria of Allen and Brown[7] which are: (1) Episodes of Raynaud's phenomenon excited by cold or emotion, (2) bilaterality of the phenomenon, (3) absent or minimal cutaneous gangrene, (4) absence of any primary causal disease, and (5) a history of symptoms being noticed for two years or longer. All presented bilateral, symmetrical, upper extremity triphasic vasospastic phenomena on exposure to cold, and showed no evidence of any other causal condition. Two of the patients were Negroes (10 per cent). The men varied in age from 23 to 42 years. The duration of symptoms varied from two to 15 years, with one individual stating that he had noticed blanching of his fingers on exposure to cold since childhood. All had involvement of both upper extremities, and four (25 per cent) had additional involvement of the lower extremities. There were no cases of simultaneous involvement of all four extremities as an initial manifestation. The duration of the disease did not seem to determine its severity, since some were rapidly progressive. Exposure to cold was the greatest initiating factor; and the critical temperature was variously reported as 57° to 60° F., at which point the vasoconstrictor phenomena would be incited.

The physical findings were minimal, although one case showed early sclerodermatous changes in the digits, and some cases presented rounded finger nails and some atrophy of the finger pads. All demonstrated adequate peripheral arterial pulsations with no gross evidence of arterial insufficiency. Careful evaluation of psychogenic factors was done. It is imperative to do this since Mufson[8] emphasizes the psychosomatic disturbance as the mechanism of Raynaud's disease, and successfully treated six cases, by eliminating these factors. Six cases presented marked neuropsychiatric disturbances requiring psychotherapy, and these were eliminated from any consideration of sympathectomy.

The vasospastic attacks were studied before, during, and after regional sympathetic ganglion block with procaine, utilizing oscillometric and thermocouple determinations before and after block, and before and during exposure to cold environment. In the upper extremity, stellate ganglion block was employed in three, and dorsal sympathetic ganglion block in two of the operative cases, and no particular advantage of one method over the other was noted. For the sake of simplicity, stellate ganglion block has been routinely used to evaluate the other cases. In every instance the resistance to cold exposure increased, and although the local response to cold, as emphasized by Lewis,[9] still could occur, recovery from the vasospastic manifestation in the blocked extremity was much swifter than the opposite one under identical conditions. It is recognized that there is an active controversy between the supporters of the conception of increased sensitivity of the sympathetic nervous system

(vasomotor theory), as advanced by Raynaud in 1862,[10] and the group favoring the local fault theory of Lewis. All twenty cases were examined and some treated during the more severe manifestations, by sympathetic ganglion block, and it is worthy of note that even the mild cases improved subsequently. Whether the manifestation of Raynaud's phenomenon is one of local fault[9, 12, 13] or a consequence of more central vasoconstrictor influence[10] is difficult to decide. It is quite reasonable to assume that the manifestations of Raynaud's phenomenon are probably a combination of the two factors. It is interesting to note that Lewis (1936)[9] recognized preganglionic sympathectomy to be more effective than ganglionectomy, for the relief of vasospasm. Some investigators[14] who support the theory of Lewis, that Raynaud's disease is primarily a local disease of the digital arteries, advocate operation, because paralysis of the vasoconstrictor nerves results in increased caliber of the denervated arteries. Local spasm, which may take place following sympathetic denervation, consequently, should be less damaging since the lumina of the vessels involved are larger. On this basis, nine preganglionic cervicodorsal sympathectomies were performed upon five of the more severely progressing cases in this group, with satisfactory results in four and partial failure in one. It is to be emphasized that out of a group of 20 such cases, 15 were treated medically with excellent results, and this is the treatment of choice in the milder cases. That the medical treatment of Raynaud's disease is not entirely satisfactory is stressed by Allen, Barker, and Hines.[15] They feel that the surgical treatment with sympathectomy still remains the most satisfactory method of treatment in Raynaud's disease. At the Mayo Clinic only progressing lesions are operated upon. Results at the Mayo Clinic in upper extremities in the early or moderately advanced cases are as follows:

1. In a small percentage of cases (10–15 per cent) complete and permanent relief has been obtained.
2. In about half, good but not complete relief has been obtained.
3. In the remainder there has been no relief, or if relief has resulted, it has persisted for only a few months or a year or two.

In the advanced cases, gratifying healing of trophic lesions has been obtained, but relief of the Raynaud's phenomenon has usually not persisted, and eventual advancement of the sclerodermatous changes has not been prevented.

One of the most optimistic reports published, concerning the surgical treatment of Raynaud's disease, is that of White and Smithwick[16] in which 93 upper extremities were denervated for primary vasomotor disorders, with good results in 65 (70 per cent). Shumacker[17] reported 26 sympathectomies on 13 patients with vasospastic diseases. Eight were in patients suffering from the common type of Raynaud's disease, and he felt that the procedure was very beneficial. Other reports have not been enthusiastic. Johnson[12] studied five cases which had sympathectomies for Raynaud's disease. In 17 to 35 days, the circulation, as tested by finger volume pulsations, returned to previous levels, although temperatures remained elevated and the absence

of sweating persisted. He does not favor sympathectomy. Fontaine, Forster, and Stephanini,[18] reporting the late results in three cases, found improvement in two following bilateral splanchnicectomy, upper lumbar sympathectomy, removal of the left adrenal gland, and extirpation of both stellate ganglia. They came to the conclusion that the disease must be associated with the autonomic function of the arterioles and capillaries. They believe that the surgical procedures are done too far away from the seat of the abnormal vasoconstrictor phenomena.

The failure of the surgical treatment of Raynaud's disease in certain cases is usually in the upper extremity, as occurred in one of my cases, and has been attributed to a variety of causes. Although there is remarkable unanimity as to the surgical procedure for lumbar sympathectomy, concerning the extent and location of resection, this is not true in the upper extremity. The crux of the matter appears to be whether the first thoracic nerve contributes sympathetic fibers directly to the stellate ganglion. This point has been raised by the studies of Kuntz[19] and his coworkers,[20] who feel, on the basis of animal experimentation and clinical observation, that the first thoracic nerve contributes sympathetic fibers directly to the stellate ganglion and the upper extremity, and following functional reorganization of pathways after preganglionic cervicodorsal sympathectomy, there is frequently failure of the operation. They are of the opinion, that the attempt of Telford[21] and Smithwick[22] to avoid adrenin sensitization of the vascular musculature in a sympathectomized extremity by the preservation of the first thoracic nerve thus retains these fibers and accounts for failures. The phenomenon of sensitization has been extensively studied by Cannon and his collaborators,[23] and has been investigated particularly in Raynaud's syndrome.[24] This sensitization is less marked if a preganglionic section is done leaving the ganglion cells with their axons intact. The view taken by Kuntz is supported by the observation of Ray, Hinsey, and Geohegan[25] who made observations of the "Distribution of the Sympathetic Nerves to the Pupil and Upper Extremity as Determined by Stimulation of the Anterior Roots in Men." Other factors to be considered are sympathetic nerve regeneration,[26] recovery of intrinsic peripheral vascular tone, the role of sympathetic vasodilator pathways, multiple arteriovenous shunts, humoral and metabolic control of the circulation through denervated vessels, abnormal spasm of the peripheral vascular bed, and the possibility that the decentralized ganglion in preganglionic sympathectomy may be the source of vasoconstrictor tonus, and thus not produce a maximal desirable result.[27]

The preponderance of surgical opinion favors the use of preganglionic sympathectomy, and this is the method that I have utilized in five cases (nine extremities) of Raynaud's phenomenon. The extent of sympathectomy can be easily determined postoperatively by the use of the electrical skin resistance determinations,[28] or the performance of a sweating test.[29] This method has been utilized frequently in the cases being reported in order to ascertain accurately the extent of the sympathectomy. The extent of denerva-

tion is uniform in the upper extremity by employing the method of preganglionic sympathectomy as reported by Smithwick.[22] The results of sympathectomy in Raynaud's phenomenon in four cases were excellent, but there was a poor result in one case in which the reaction to cold was still severe, although there was a more rapid return to normal after exposure to cold on the sympathectomized than the unsympathectomized side. It is felt that a conservative attitude toward the surgical treatment of Raynaud's disease is in order, and that sympathectomy should be employed only in the severe and progressive cases, particularly with early sclerodermatous and ulcerative changes.

ATYPICAL CAUSALGIA

There were six cases in this group worthy of consideration, upon whom one cervicodorsal and five lumbar sympathectomies were done. Despite enthusiastic reports to the contrary,[29, 30, 32, 33] I have not been impressed by the results in lesions in the lower extremity associated with edema. The cases of true causalgia in which the discomfort is limited to the anatomic distribution of the involved nerve[31, 34] were excluded. One is impressed by the disappearance of many of the painful manifestations, and where the lesion appears to be one principally of vasoconstriction with sensitivity to cold, the response is excellent. I have had occasion to see a considerable number of unilateral lymphedemas of the upper extremity with extreme tenderness of the extremity. A case which responded dramatically to sympathectomy is as follows:

(A. S. C.) A white man, age 26, was exposed to poison oak and developed sufficient cutaneous reaction to require hospitalization. After the dermatitis had subsided, he noted his right hand had become stiff, and soon thereafter the fingers became cold, swollen, painful, and tender. He received hot soaks, physiotherapy, and whirlpool, as well as contrast baths and massages, but to no avail. He was first seen by me three months later with evident pitting edema of the entire hand and fingers, marked mottling of the skin, and trophic changes in the fingernails. He experienced considerable pain on exposure to cold, and there was marked tenderness to touch. There was no clinical evidence of arterial insufficiency, and the oscillometric readings were equal at the wrist. Roentgen-ray films of the hand showed coarsening of the bony trabeculations. He was treated unsuccessfully as regards the primary findings, over a prolonged period by elevation, compression, physiotherapy, and active exercises. Neuropsychiatric evaluation ruled out a major psychosomatic factor. On exposure to *cold,* it was noted that the cyanosis of the skin became greatly exaggerated, and this was well controlled temporarily by stellate sympathetic ganglion block. Since the results were only temporary, a cervicodorsal preganglionic sympathectomy was done. This resulted in dramatic disappearance of the pain, swelling, and cyanosis. There was also gradual improvement and finally disappearance of the stiffness of the fingers. Subsequent follow-up, one year later, showed the individual to be completely rehabilitated.

This case demonstrates the effectiveness of sympathectomy in ameliorating these atypical causalgic manifestations, following almost insignificant trauma, which if allowed to progress will result in irreparable and almost complete disability. Great care must be taken to evaluate any psychosomatic

component which might account for the unilateral lymphedema. This has been observed as a hysterical manifestation, which responded completely and immediately to narcosis and psychotherapy. These cases, however, usually refuse to move their extremity, or allow it to be touched, and have edema of the entire extremity, whereas the reported case presented a localized type of swelling.

Obviously all cases presenting marked vasospastic phenomena do not require sympathectomy. A man (S) aged 30, white, was seen, complaining of coldness and paresthesias of the left lower extremity. A ruptured meniscus had been excised from the left knee four months previously. The left foot was colder than the right, and the peripheral arterial pulsations were slightly diminished on the involved side. The left knee joint appeared satisfactory, and there was no evidence of a primary nerve lesion or thrombosis. Examination of the extremity under standard conditions of temperature (68° F.) and humidity (50 per cent) demonstrated that the left foot was 7° cooler than the right. Left lumbar sympathetic ganglion block produced immediate rise of temperature to the extent of 18° F. All the symptoms promptly disappeared, and there has been no recurrence of the condition. This obviously represents a case in which there was a primary vasospastic phenomenon which responded satisfactorily to lumbar sympathetic ganglion block.

The majority of cases followed minor trauma, such as a rather insignificant fracture, minor shrapnel wound, or exposure to environmental trauma (dermatitis). All responded excellently to sympathectomy except those associated with lymphedema of the lower extremity. In two cases in this group, both following fracture of malleoli with prolonged incapacitation before being seen, lumbar sympathectomy was unsuccessful in controlling the edema, and in one case, the pain. The mechanism of this failure must be related to a prolonged state of vasospasm resulting in persistent edema and finally fibrosis with a more or less fixed edema. One patient presented ulceration over the malleolus, which healed following the sympathectomy, but the edema did not subside, and both patients are still incapacitated. This points to the fact that these cases must be operated upon early to achieve a satisfactory result. In the late stages, this edema can be controlled by elastic support and elevation.

The mechanism of these sequelae to trauma have been variously explained by the concept of the internuncial pool[33, 35] and the Lovén reflex.[36, 37] The concept of the internuncial pool as advanced by Lorente de Nó[35] and adopted by Livingston[33] is based on the premise that a prolonged bombardment of painful impulses sets up a vicious cycle of reflexes spreading through a pool of neuron connections. Because of the summation principle of nerve impulses, there is kept alive within such a pool a constant circling of activity across the synapses involved. The afferent pathway is represented as the sensory nerve fibers traveling in the posterior root. As a consequence, the abolition of pain and vascular spasm and its sequelae results from the interruption of the efferent sympathetic pathways leading from the pool when ganglion block or sympathectomy are employed. The vicious reflex is thus interrupted with beneficial results.

SUMMARY

1. Experiences with ten cervicodorsal and seven lumbar preganglionic sympathectomies in five cases of Raynaud's disease, six cases of atypical causalgia, and two cases of cold sensitivity and pain after ligation of main vascular channels are related.

2. The feature of sensitivity to cold, which is a predominant manifestation of vasospastic disorders, is emphasized. Eighty per cent of patients requiring sympathectomy, in the author's experience, have presented this as an initial and predominant manifestation. Certainly, primary vasospastic conditions should demonstrate this more frequently than any other vascular disease.

3. One should not teach that Raynaud's disease rarely occurs in men. A group of 20 cases in men with typical manifestations are reviewed.

4. The results of preganglionic sympathectomy in Raynaud's disease have been excellent, with a poor result in one case. Surgical treatment is desirable only after careful evaluation and only in severe progressive manifestations.

5. Sympathectomy for atypical causalgic manifestations has resulted in excellent recovery except where the lesions were associated with edema of long standing in the lower extremity.

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MULTIPLE INTUSSUSCEPTIONS, DIRECT AND RETROGRADE, OF TRAUMATIC ORIGIN

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ACUTE INTUSSUSCEPTION commonly is a disease of infancy, however, it may occur at any age and as Miller[1] states it "may develop at any level in the alimentary tract, may be single or multiple, descending or retrograde in type, and have as its basis a wide range of pathologic conditions—." Multiple areas of intussusception, retrograde intussusception, and traumatic intussusception are each a rarity; the combination of these lesions in one case forms the basis for this case report and review of the literature.

I. SOLITARY AREAS OF RETROGRADE INTUSSUSCEPTION

Groper[2] classified retrograde intussusception according to the area of intestine involved. He included (1) jejunogastric, (2) enteric, (3) ceco-ileal, and (4) colic intussuception. Bauman[3] states that the ratio of retrotrade to direct intussusception is 1:200.

A. Jejunogastric Intussusception. Though gastro-enterostomy has been performed since 1881 the first case report of retrograde intussusception of the jejunum into the stomach did not appear until 1917. (Adams, 1935).[4] Since that time Becker,[5] Debenham,[6] Drummond,[7] and others[8] have reported cases. Though the etiology remains obscure no cases of traumatic origin have been recorded, and so the entity will not further be considered in this report.

B. Enteric Intussusception. Ibos and Legrand-Desmons,[9] Ladd,[10] Caminiti,[11] and many others have reported cases of solitary retrograde enteric intussusception. Homans[12] reported the following interesting case of recurrent retrograde intussusception in an Armenian girl. On initially exploring her the process "was easily reduced," however during her hospital stay she suffered recurring episodes of abdominal distress similar to that experienced prior to surgery. He continues, "She was thin and had a long mesentery so we advised her to fatten up if possible." The patient gained weight but returned in six months with similar complaints and a "pulse not over 90 and a high white count, vomiting very little, the bowels moving every day." She was observed and finally reopened by Doctor Cheever who found "an intussusception in the upper intestines which he was forced to resect." There was no tumor, and both the initial and the recurring intussusceptions were in a retrograde direction. Doctor Homans concluded "There seems to be no possibility of preventing such a strange condition."

Mitchell[13] reported the successful resection of a gangrenous retrograde ileo-ileal intussusception that was found in a 15-year-old girl. Symptoms of two days' duration preceded the operation, and no tumor or other abnormal local condition was found to explain the lesion.

Recently, Lannon[14] and Culiner reported a retrograde intussusception in a ten-month-old malnourished child. The duodenum, pylorus, and lesser curvature of the stomach were invaginated into the lower esophagus, and death resulted from the obstruction.

C. Ceco-Ileal Intussusception. Of Groper's[2] 4 classes of reverse intussusception ceco-ileal apparently is the most rare. Thorek and Lorimer[15] performed a right hemicolectomy for a retrograde ceco-ileal intussusception. Carcinoma of the cecum invaginated into the dilated, chronically obstructed ileum; resection was followed by recovery.

McSwain[16] reported a case of retrograde intussusception of the appendix, so that apparently any of the mobile segments of the gastro-intestinal tract may be the site of this process.

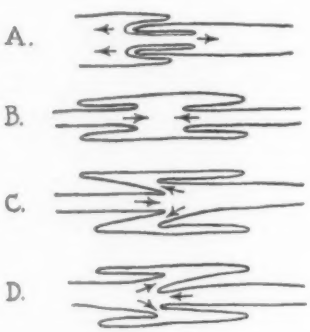
D. Colic Intussusception. Balfour[17] in 1918 described a retrograde intussusception of the sigmoid colon. A pedunculated malignant papilloma was found at the apex of the intussuscipiens. Balfour then observed the reformation of the retrograde intussusception as "the tumor was again drawn upward by powerful antiperistaltic contractions of the proximal sigmoid, these contractions extending upward for a distance of from 12 to 14 inches above the site of the tumor." "The process of invagination, begun in this manner, continued until the portion of the bowel containing the tumor was drawn upward and completely engulfed by the proximal segment."

Sussman[18] reported a case in which a lipoma of the splenic flexure was at the apex of a retrograde intussusception. In Lewis'[19] case a 20-inch long pelvic mesocolon was indicted as allowing an extensive sigmoid colon invagination. Schoenfeld[20] reported a similar case in a four-month-old child. Fleming[21] and Lazarus[22] have also reported cases of colic retrograde intussusception.

II. Multiple Intussusceptions, Direct and Retrograde. Multiple areas of intussusception were present in but 12 of the 1000 cases reported by Fitzwilliams, [23] and in but three of Ladd and Gross'[10] 372 cases. Kahle[24] in a series of 151 cases reported three instances: one case had six separate enteric and one ileo-cecal mass, and another case in a 21-month-old infant had two separate areas of retrograde intussusception. Gill[25] reported that in a 19-month-old female child "six or seven separate ileo-ileal intussusceptions were found close together"; the proximal invagination caused complete obstruction and was the only area to show signs of bowel wall congestion. Baron[26] found five separate areas of intussusception in a two-year-old boy. These areas averaged 1.3 cm. in length and occurred within a 25 cm. length of jejunum. Badertscher,[27] Le Conte,[28] and others[29-38, incl.] have reported single cases; one of Todyo's[39] three cases is unique enough to deserve description. A 20-year-old Japanese man with a strangulated inguinal hernia was operated 12 hours after the onset of symptoms. Resection of gangrenous intestine 121 cm. above the ileo-cecal valve was performed, and further examination revealed ten separate areas of intussusception in the proximal intestine. Most of these were retrograde and some were superimposed; they were reduced without difficulty, and the patient was discharged on his 16th postoperative day. Twenty-four

days later severe abdominal pain recurred and celiotomy revealed that the lower end of the ileum was strangulated by a band. The band was severed; "at this time multiple intussusceptions were again found in different portions of the small intestine, the uppermost one, 155 cm. below the duodeno-jejunal flexure, showing a retrograde, and usual (downward) intussusception at the same time." Todyo continued "Four others were found in a very early stage of development of an invagination, each showing a ring-like fold of the intestinal wall (Fig. 1, b.). They were all reduced."

III. Contiguous Direct and Retrograde Intussusception. The varieties of contiguous direct and retrograde intussusception are illustrated in Figure 1.[40] Other variations in the living are possible, however, none were found in the literature. The entity was titled "reverse type intussusception" by Brocq,[41] and "double intussusception" by Mitchell[42] and Hipsley;[40] both of these terms are loose, and in this report 'contiguous direct and retrograde intussusception' is used collectively to describe the types illustrated in Figure 1. Clubbe[43] very briefly mentions a variety of such a combination in a child in whom an ileocecal invagination upon reaching the descending colon appeared—"to form a retrograde movement *en masse* and to be invaginated into the colon higher up the bowel. So, in reduction, pressure first in one direction, then in the opposite, was necessary . . ." (Fig. 1, a.)



(A.B.C. redrawn from "Intussusception" by P.L. Hipsley, M.D.)

FIG. 1.—Contiguous direct and retrograde intussusception. A. Initial direct intussusception (to the right) then passes in a retrograde direction. B. Variety found in case herein presented. C. The retrograde portion enveloping the direct process. D. Similar to C, except for the inclusion by the direct of a portion of the retrograde.

Catz[44] reported several cases similar to that described by Power (Fig. 1, c) as well as cases in which the direct and retrograde intussuscipiens met head on, as in the case reported later in this article, (Fig. 1, b).

Buckley[45] reports the occurrence in a two-year-old child of a direct ileocecal intussusception having then invaginated into an adjacent retrograde intussusception of the transverse colon, Fig. 1, c. Redundancy of its mesentery allowed the cecum to be placed in any abdominal quadrant. Wells[46] reported two similar cases in which the ileocecal intussicipiens had progressed so as to be felt at the anus. A reverse intussusception of the sigmoid colon outside the former mass was found to extend to the splenic area of the mesocolon. D'Arcy Power's[47] case had, in addition to the envelopment of the direct intussusception by the retrograde process, the entire mass in turn recessed into a second retrograde intussuscipiens. He concluded that "The third invagination was

much smaller and was formed after death by a wrinkling of the bowel." The direct process in this case measured three and a half inches and the retrograde two and a half inches. Power adds: ". . . I believe it to be an example of an intussusception which is by no means uncommon. It is a particularly deadly variety, first, because distention of the colon has no effect upon it, and secondly, because after a laparotomy, any attempt to reduce the intussusception tends to increase the retrograde invagination and so make matters worse . . ." Moutard Martin,[48] Knaggs,[49] and Ryan[30] report similar cases.

In the cases of Kleberg[50] and of Sainet[51] the descending intussuscipiens enveloped the retrograde mass (Fig. 1, d).

IV. Traumatic Intussusception. Leichtenstern[52] in 1873 reported a series of 326 cases of intussusception in whom there was available a thorough history; of these 26 or 8 per cent had a definite history pointing to trauma as the etiologic agent. He stated further that "The first symptom of the intestinal invagination usually followed the traumatic effect immediately." In the 26 cases contusions of the abdomen accounted for 14, and concussions or severe physical exertion was indicated in the remaining 12. Eliot,[53] Haun[33] and others[54-57, incl.] have contributed reports in which a sudden increase in intra-abdominal pressure has apparently caused an intussusception. Hipsley[39] reported 100 cases of intussusception and stated that "several of the cases . . . began immediately after a fall out of a perambulator . . ."

Instances of multiple areas of intussusception following trauma have also been described. Le Conte[28] in 1898 treated a nine-year-old boy who had been stabbed in the left side of the abdomen. Celiotomy revealed a direct jejunal intussusception about one inch long. About two feet distal to it two additional invaginations were found, one direct and the other retrograde; each was about ¾ inch long (Fig. 1, b). No signs of inflammation, congestion, or change in the color of the intestine were present, and reduction was accomplished by very light traction. W. C. Peters[36] found three separate areas of intussusception in an eight-year-old boy who had suffered an abdominal injury in an auto accident. Badertscher[27] reported a similar case in a nine-year-old boy whose lower abdomen was run over by two wheels of a truck. This child had three typical areas of intussusception.

Case Report.—G. E. N., technician 5th grade, 104th Infantry Division, vicinity of Duren, Germany, January 9, 1945, at 6:30 P.M. while manning a jeep-mounted 30-caliber machine gun, was struck in the left lumbar area by a fragment of an aerial bomb that detonated some 10 yards away. Presumably the bomb was a 200–250 Kg. anti-personnel missile, and the effect of the combined blast and shell fragment was to knock G. E. N. from his weapon. He estimated that he was able within a matter of several seconds to rise and aid 2 mortally wounded comrades to places of safety some 50 feet distant. He subsequently collapsed and during the next 8 hours' evacuation received 2 units of plasma in both the collecting and in the clearing stations. On admission to the Shock Ward of the 53rd Field Hospital, 2nd Hospital Unit, Eschweiler, Germany, he was found by Captain J. O. Price to be in moderate shock and in the next 30 minutes was given 1000 cc. of blood. Physical examination revealed a 1 cm. in diameter per-

forating wound of the left upper lateral gluteal area with a 3 cm. in diameter wound of exit at the left pubic tubercle. This latter area was filled with blood clot. Circulation of the left lower extremity was normal. There was no evidence of a "blast injury" to the eyes, ears, or lungs (physical examination and roentgen-ray). Roentgen-ray revealed a compound fracture of the left acetabulum and pubis. At 3:30 A.M. (9 hours post-injury) on January 10, under intratracheal gas-oxygen-ether anesthesia (administered by Capt. Alvin Leonard) a left lower rectus-splitting celiotomy was performed by Major L. L. Hall and myself. A severe laceration of the sigmoid colon, an incomplete transection

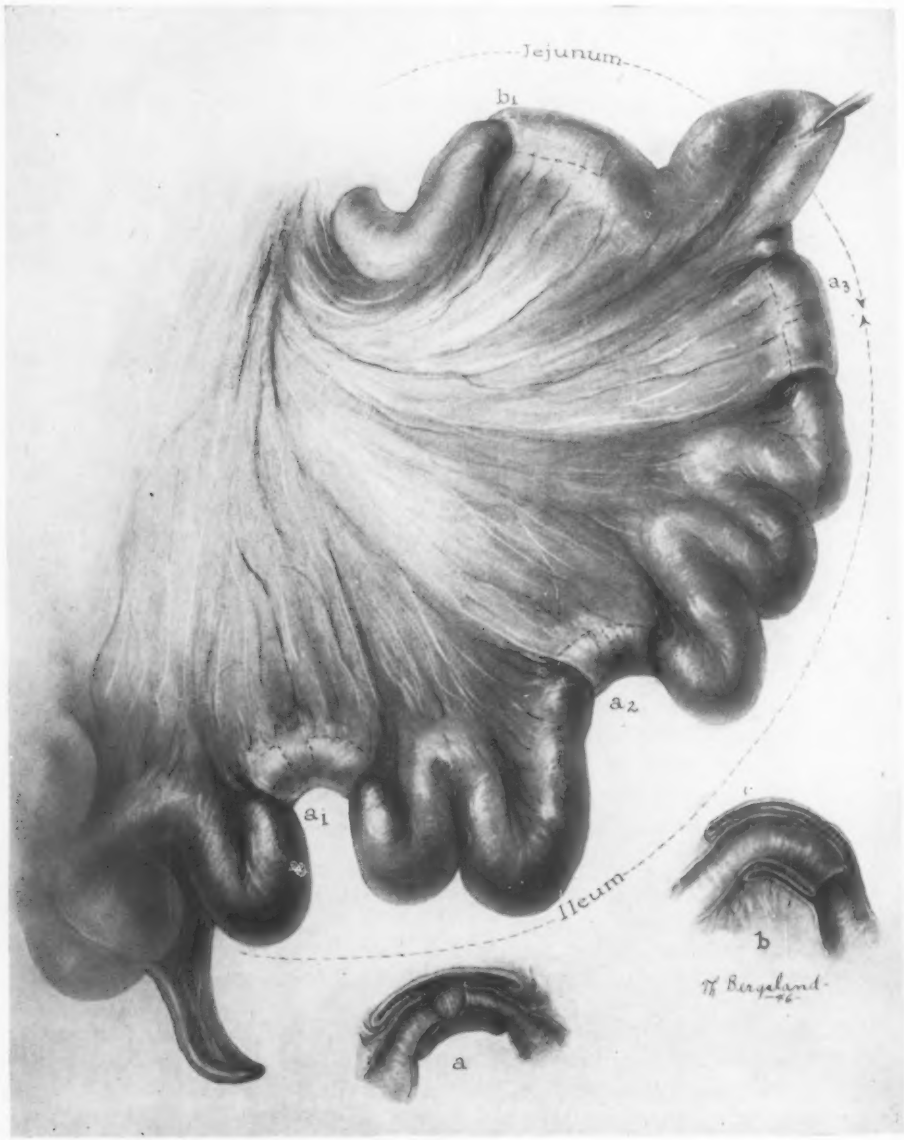


FIG. 2.—Multiple areas of direct and retrograde intussusception. A. Sagittal section of type of invaginations found in areas a_1, a_2, and a_3. B. Classical intussusception: sagittal section of invagination found in area b_1.

of the external iliac vein, and a transection of the inferior epigastric artery were discovered, and these lesions were repaired. The gastro-intestinal tract was then further examined. Located in the mid jejunum was a 3.5 cm. long area of intussusception (Fig. 2). Lower in the jejunum and in the ileum were 3 areas of a Fig. 1, type b, contiguous direct and retrograde intussusception. The invaginated areas each measured about 2 cm. in length and were milked out with a moderate degree of difficulty. A transitory slight blanching of the intussuscipiens was noted. There was no peristaltic effort made to reform these areas nor was there any apparent abnormality in the small intestine. The appendix had been removed in 1937, and the patient stated that the only abnormality found at that time was that his appendix "was behind the colon."

Convalescence was uneventful, and the patient was evacuated on his 17th hospital day. His colostomy was subsequently repaired and when heard from on April 10, 1947 he was in good health, had had no recurrence of any gastro-intestinal disturbance, and was attending college. Never in his life had there been signs or symptoms suggestive of a disturbed autonomic nerve balance.

DISCUSSION

It is held that trauma was the etiologic agent in this instance of multiple area of direct and retrograde intussusception. Presumably in this case the nerve tension of battle caused a violent central (Bockus[58]), and adrenergic stimulation of the sympathetic nerves of the gastro-intestinal tract with a resultant spasm of the sphincters and segmental spasm of bowel (McSwiney[59]). The abdominal wall blast, though the positive wave was applied to the patient's side and to his back, gave rise to enough increased peritoneal cavity pressure to drive many spastic areas into adjacent areas of relative dilatation. The effect of the fall to the floor of the jeep would have the same effect. Of more than academic interest, too, would be the knowledge of what is the effect of some of the more highly seasoned of the K and C rations on the intestinal motility; this patient was injured only a few minutes after a meal of one of those stock front line rations.

One might well question why, if of traumatic origin, this should be the only case report to be found in the voluminous literature welling from this War; this was the sole instance of such a finding in our own review of 1,063 acute war wounds of the abdomen and chest.[60, 61, 62] Thus, it must be admitted that more factors than trauma alone are involved, viz.; the factors of a recently ingested meal, severe fright, and the still possible existence of some autonomic nerve imbalance in the soldier. Yet, it seems apparent that without trauma as the trigger mechanism these other factors never would have produced the remarkable lesions.

CONCLUSION

A review of the literature and a case report is presented of multiple areas of direct and retrograde intussusception of traumatic origin.

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CARCINOSARCOMA OF THE UTERUS*

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CARCINOSARCOMA has been reported in many organs but the site of predilection is the uterus. Robert Meyer[1] divides it into three groups: (1) collision tumor, in which a carcinoma and a sarcoma arise at different sites and fuse by growth contiguity; (2) combination tumor, in which the two malignant elements are derived from one stem cell, as in the Wilms tumor; (3) composition tumor, in which parenchyma and stroma of a single tumor become neoplastic. Jaffé[2] would limit the term carcinosarcoma or sarcocarcinoma to the last group and it is so used in this communication.

The carcinosarcomatous nature of uterine tumors has been questioned by several observers. Saphir and Vass[3], in a review of 36 cases from the literature, stated that perhaps three or four may possibly be so designated but do not fully accept them. Outerbridge[4] believed that there was no such entity as "carcinosarcoma." Willis[5] in 1924, stated that in human pathology, there was no acceptable example of a sarcomatous change in the stroma of a tumor. Pitfalls in the diagnosis have been pointed out by many observers, the chief one being the marked polymorphism of carcinoma cells[3, 4, 6, 7] which, when anaplastic, can closely resemble sarcoma cells. Saphir and Vass also stress other features which may cause difficulty—chronic inflammatory cells in the region of the tumor, a history or histologic evidence of irradiation therapy, and the fact that of the reported cases, none have had metastases showing the combined feature; the malignancy has been either pure carcinoma or pure sarcoma.

The development of carcinosarcoma is explained by Jaffé as due to three possible mechanisms: (1) the primary tumor is carcinomatous and the stroma develops sarcomatous features; (2) the sarcoma is first and is followed by carcinomatous changes; (3) both blastomatous elements develop simultaneously. Ewing[8] believed that at the point where a sarcoma reaches the endometrial surface, carcinoma could develop secondarily or that a common irritant could produce neoplastic changes in both elements. It is well known that such tumors occur in animals both spontaneously and under experimental conditions. Our interest in carcinosarcoma of the uterus was aroused by the following two cases which came under observation.

* Since this article was written, the report of Stein (Monatschr. f. Geburt. u. Gynäk. **36**, 417-438, 1912) has become available. He reported a case of a 46-year-old woman with adenoacanthosarcoma, associated with adenomyosis of the uterus and having extensive sarcomatous abdominal metastases.

CASE REPORTS

Case 1.*—The patient, a white woman 63 years old, was first admitted to Doctors Hospital on July 31, 1945, because of metrorrhagia of three months' duration. Menopause had occurred 10 months previously. Since the onset of the current illness, there had been periodic spotting and bleeding, without any weight loss. The only other features of note were chronic constipation, slight exertional dyspnea, palpitation, ankle edema and frequent headaches.

Examination revealed an elderly obese woman, not acutely ill, whose heart was slightly enlarged with a soft systolic apical murmur, a blood pressure of 190 systolic and 110 diastolic and a slight albuminuria. Otherwise there was nothing of note.

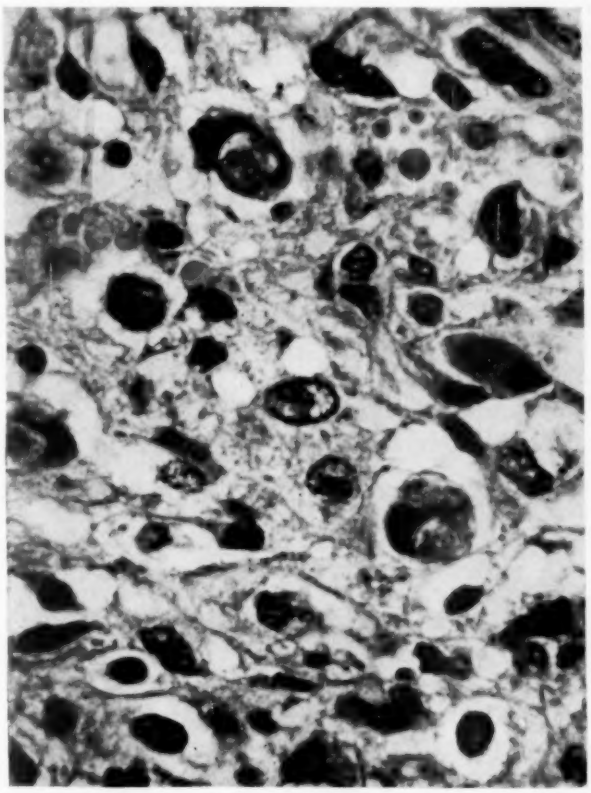

FIG. 1.—Case 1: Retroperitoneal sarcoma. × 600

Curettage gave considerable amounts of papillary cauliflower tissue, obviously malignant, so hysterectomy was performed. The histologic diagnosis of endometrial carcinoma was made by one of us (JRL). The uterus was enlarged and distorted by many intramural fibroids, the largest 3 cms. The endometrium showed only the changes seen after curettement. The fibroids showed only the usual changes; there was no evidence of malignancy. During the postoperative course, pyuria and albuminuria developed. She was discharged August 12.

* Thanks are due to Dr. John H. Garlock for the clinical data of this case.

About six weeks after leaving the hospital, signs of peritoneal irritation developed and a mass in the right lower quadrant which rapidly increased in size. There was no weight loss. She was readmitted to the hospital on December 12. A large hard, nonpainful, nontender mass about the size of a fetal head was palpable in the lower abdomen, apparently semifixed and somewhat globular. There was a moderate anemia.

At operation, a large retroperitoneal mass filled the right lower quadrant, was bluish in color and consisted of grumous material with soft white masses of fish-flesh appearance. There was excessive bleeding. Death occurred January 1, 1947. Autopsy was not obtained.

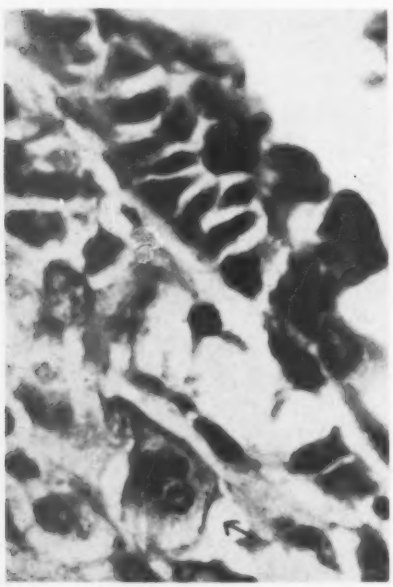

FIG. 2.—Case 1: Curettings showing atypical malignant epithelial surface cells and atypical stromal cells with atypical triangular mitosis, indicated by arrow. × 600

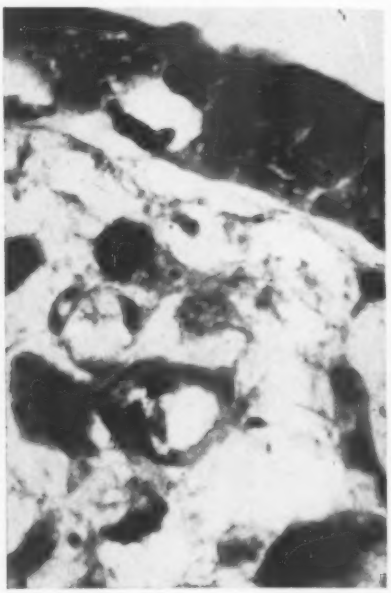

FIG. 3.—Case 1: Curettings showing the single layer of carcinomatous cells and the atypical hyperchromatic stromal cells. × 600

Microscopy of the retroperitoneal mass showed typical sarcoma (Fig. 1). Re-examination of the previous curettings revealed a feature which had been overlooked. In addition to the carcinomatous epithelium, the stroma consisted of atypical cells showing marked variability in size, hyperchromatism of nuclei and atypical mitoses (Figs. 2 and 3).

Final Diagnosis.—Carcinosarcoma of the endometrium with retroperitoneal metastatic sarcoma; multiple fibromyomas of uterus.

Case 2.—The patient was a 54-year-old white woman of U. S. nativity admitted to Goldwater Memorial Hospital, service of Dr. Condict W. Cutler, Jr., on January 24, 1946, because of vaginal bleeding. The current illness began suddenly four days previously with lower abdominal cramps followed by vaginal bleeding increasing in severity until a few hours previous to entering the hospital.

Menses began at 9, occurred every 20 days and lasted for five days with a moderate flow. There had been two normal pregnancies. At 44, there were periods of vaginal bleeding lasting for weeks, interrupted by a few free days. An artificial menopause was induced with radium. During this year and the next, there were attacks of prolonged

vomiting of undetermined origin. Until the age of 47, vaginal bleeding ceased, to reappear two to three times annually for the next three years. The bleeding episodes were painless; discharge had never been noted. Until the current illness, she had remained asymptomatic.

There was a past history of mild cardiac failure, a moderately high tension for many years and attacks apparently of gallbladder disease with right upper quadrant pain and marked idiosyncrasy to fatty foods. At 27, she had an appendectomy and right ovariectomy and at 31, pneumonia, empyema and a period of observation at Saranac. At 43, the gallbladder had been removed. During the last few months, there was vague costovertebral angle pain and some weight loss.

Physical examination showed a plethoric, intelligent, white woman. The right lower quadrant was tender, had slight spasm on deep palpation and a sense of fulness but without definite mass. Vaginal examination showed a normal cervix. Further gynecologic examination was not carried out for fear of precipitating further bleeding. The

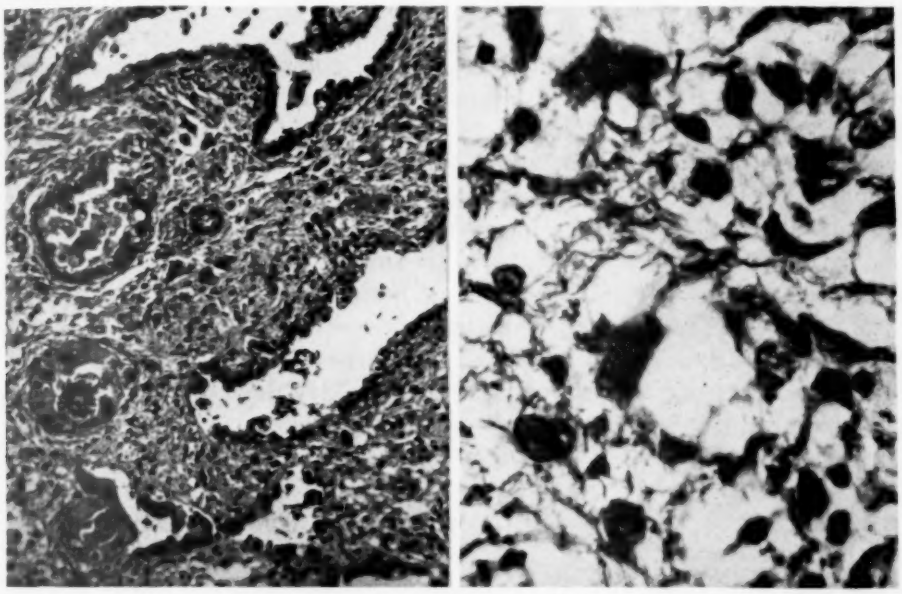

FIG. 4.—Case 2: Endometrial surface showing glands lined by atypical cells and deeper areas with malignant squamous metaplasia. One gland has both types of malignant cells. × 90

FIG. 5.—Case 2: Endometrial stroma with large hyperchromatic macronuclei. × 400

general examination showed a heart with an apical systolic murmur, an accentuated second aortic sound, no evident enlargement, blood pressure 200 systolic and 105 diastolic. Clotting time was 3 min., bleeding time 1 min. Other laboratory tests showed nothing of note.

Laparotomy was performed on January 25. A large semicystic boggy uterus about the size of a three months' pregnancy was found and removed supracervically. The stump was quite friable.

The postoperative course was uneventful and she was discharged on the 17th hospital day.

Pathology Report.—The uterus, 9 x 6.5 x 9 cms. is removed supracervically with the left tube. On opening it contains a degenerating semigelatinous gray-brown mass which is adherent in only a few areas and generally separates with ease, leaving a relatively irregular firm surface. The wall measures 1 cm. in thickness.

Microscopy.—The endometrial glands and surface are lined by neoplastic tall columnar cells. The glands penetrate deeply into the wall, where they preserve their architecture and have pronounced papillation. In the endometrium they tend to be somewhat scantier than usual and have transitions to malignant squamous cells (Fig. 4). Striking changes are present in the endometrial stroma. There are many small hemorrhages, some of which enclose greatly dilated capillaries. The stromal cells (Fig. 5) tend to be large, many of the nuclei are hyperchromatic, of increased size, have macronucleoli and bizarre mitoses. There are several large nests with giant nuclei. The stromal cells are in sharp contrast with the epithelial cells. Reticulum stains show fine fibers; in the large nests, they are extremely abundant. Cells similar to those of the surface endometrial stroma are not found within the myometrium.

Diagnosis.—Carcinosarcoma of the endometrium.

Gebhardt[9] in 1899 appears to have reported the first case of carcinosarcoma of the uterus. Meyer, after a personal examination of the slides, accepted it as authentic. Frankel[10] in 1901, reported the second case. The patient, 58 years old, for several months had profuse vaginal discharge which occasionally was bloody. The curettings were malignant and hysterectomy was performed. There were several myomas, the largest showing fibrillar sarcomatous cells enclosing papillary carcinomatous glands. The remainder of the endometrium had areas of hyperplasia, atrophy or granulation tissue. Ten months later the discharge recurred and a fungating tumor developed in the scar. She died of peritonitis a few weeks later. Autopsy revealed a necrotic carcinoma of the ovary adherent to the bowel which had perforated. The patient of Bernstein[11] was 52 years old. Uterine bleeding, more or less constant, reappeared at 48, 14 years postmenopausal. Curettage two years later revealed carcinoma but operation was refused. Panhysterectomy was performed two years afterwards, following an acute episode of intense lower left-sided pain and five weeks of continuous vaginal bleeding with foul discharge, weight loss and anorexia. During operation, the uterus tore loose from a very friable cervix and a metastatic mass was found in the omentum. The uterus was enlarged and filled with friable gray material infiltrating the wall. The endometrium after cleaning, appeared ulcerated. There was a diffuse invasion by carcinomatous glands and in some areas, closely associated with the carcinoma, was myosarcomatous tissue showing myofibrils and intercellular collagen; these areas were free of necrosis. The author interpreted the sarcomatous changes as reactive to long standing carcinoma. Klee[12] reported the case of a 58 year old woman who, six years previously, had an artificial menopause produced by x-ray therapy for hyperplastic polypoid endometrium proven by histological examination. The current illness was characterized by uterine bleeding and the curettings were diagnosed sarcoma. Panhysterectomy was performed. The endometrium showed both carcinomatous glands with columnar, cuboidal and squamous cells and neoplastic stroma having giant cells and intercellular fibrils. The patient was well 1½ years later. R. Meyer's case[13] had carcinomatous and sarcomatous changes of the endometrium. Horâlek[14] reported a case of a 56-year-old woman. Menses were regular to the age of 50, then menorrhagia developed, lasting 14 days. During the last three months, bleeding was continuous and there were several severe hemorrhages. The

uterus was enlarged, moderately soft and had a cherry sized polyp protruding from the cervix which showed histologically carcinomatous endometrial glands buried in sarcomatous stroma. Hysterectomy was performed. The endometrium was diffusely involved by polypoid growths of similar character to that previously examined. The patient was well three years later. The patient of Daniel and Lazaresco[15], 48 years old, one year after menopause, developed metrorrhagia followed by increasingly sanguinolent fetid discharge. The uterus was enlarged to the size of a six months' pregnancy. Total hysterectomy was performed. A peach sized polypoid mass arose by a broad base from the anterior wall, its surface fish-flesh in color. There were atypical papilliferous glands intermingled with atypical stroma. Various parts showed variable degrees of admixture. Harvey and Hamilton[16] reported two cases. One was an adenomyoma with carcinomatous and sarcomatous changes. The second was a carcinoma surrounded by malignant stroma. They believed that the stromal neoplasm developed secondarily to the carcinoma. Dixon and Dockerty's[17] patient was 34 years of age and two years postmenopausal. Four years previously she had received a half menopausal dose of radium following curettage, the diagnosis histologically of "endometritis" having been made. Vaginal bleeding and intermittent right lower abdominal and lumbar pain occurred a few weeks before she came under observation. A polypoid mass resembling placental tissue protruded through the os. Tissue removal by curettage was diagnosed sarcoma. The uterus, removed in toto with the adnexa, had a sessile polyp arising just above the internal os and showed papillary carcinomatous glands surrounded by neoplastic stroma. There were many benign fibroids. The patient of Barnes[18] was 48 years old. The onset of illness was three years postmenopausal with watery leucorrhea for six months and increasing spotting for six more months. Curettings were sarcomatous. Panhysterectomy was performed. There was found a local endometrial mass showing both sarcoma and papilliferous adenocarcinoma. A separate myoma was present.

The clinical symptoms of carcinosarcoma of the uterus have no distinctive features different from any other malignancy. Most of the cases have occurred after the menopause, whether spontaneous or artificially induced. In some, the sarcomatous change appears to have developed subsequent to the carcinoma, as is suggested by the case of Bernstein. In all, the sarcoma has been of endometrial origin. It is of interest to speculate on the role of radiation therapy in the development of this type of tumor. In the cases of Klee, Dixon and Dockerty and our case 2, this type of therapy had been used. Our knowledge of the late effects of minor therapy in the human is scanty and it is possible that, in susceptible individuals, malignant changes may take place after a relatively long period.

SUMMARY AND CONCLUSIONS

The literature on carcinosarcoma of the uterus is reviewed. Eleven cases are accepted as authentic and two more are added. All 13 are of endometrial

origin. All occurred after the menopause. The clinical symptomatology presents the usual features of uterine malignancy. Three cases had irradiation therapy previous to the development of the neoplasm and a possible causal relation is suggested. The sarcomatous change apparently may precede, follow or be coincidental with the carcinoma.

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CONGENITAL ABSENCE OF THE GALLBLADDER WITH CASE REPORT

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CONGENITAL ABSENCE of the gallbladder is a rare anomaly. Gross[1] in a review of the literature in 1936 was able to find only 38 cases reported since 1905. Since 1936, 13 additional cases[2-7] have been reported. Dixon and Lichtman[8] in 1945 added ten cases from both operative and necropsy records of the Mayo Clinic. This anomaly is probably not as rare as these reports would indicate. Undoubtedly many cases are encountered, which are simply not recorded. A truer estimate of the incidence of congenital absence of the gall bladder will be reached only if all cases are reported. This will also help in focusing attention on certain surgical aspects of this condition; as well as aid in the study of the patho-physiologic changes which take place in the post cholecystectomy state.

Only the 60 cases reported since 1900, which are available to us, will be considered in this review, since many of the cases reported previous to that time are lacking in details and found unsatisfactory for analytical study. Cases of congenital absence of the gallbladder, associated with atresia of the extrahepatic ducts will not be considered since they concern an entirely different problem. A case encountered by us will be presented.

The causes listed for this anomaly are many. Two, however, stand out. Both of these deal with the embryologic development of the liver and bile ducts. The two theories are as follows: (1) The hepatic diverticulum from the foregut forms the liver, gallbladder and extrahepatic bile ducts. The gallbladder and cystic duct form an outpocketing from this diverticulum. Failure of development of this outpocketing would cause an absence of gallbladder and cystic duct.

(2) The gallbladder, hepatic, cystic and common ducts in their early embryologic development are hollow structures. In the so-called solid phase, their lumina become obliterated. Failure of the gallbladder and cystic duct portions to recanalize, would cause an absence of these structures.

The condition is more common in women than in men, with 38 cases found in women and 23 in men. One report did not mention sex. This falls in line with other hepato-biliary diseases. The average age of the patients was 46 years.

In 26 of the 60 cases, the condition was found at necropsy. In not one of these cases was the cause of death hepatic or cholecystic disease. These patients apparently had no symptoms referable to this anomaly. Symptoms suggestive of cholecystic disease were present in 37 cases. Jaundice was present in 30 cases.

In 17 cases cholecystographic studies were carried out and failed to reveal a gallbladder. The diagnosis of nonfunctioning gallbladder and cystic duct obstruction was frequently made. In no case, to our knowledge, was this anomaly diagnosed preoperatively.

The gallbladder fossa in the liver was present in seven cases and absent in 27. In the others it was not mentioned. The common duct was dilated in about 50 per cent of cases in which the size of the duct was mentioned. Gall stones were found in the common or hepatic ducts in 18 cases, not found in 19 cases and not mentioned in 25 cases.

FIG. 1.—Artist's drawing of findings at operation. Wide fibrous band, holding duodenum up to hilar region of liver. Hepatic and common ducts readily visualized, with stone in the latter. No cystic duct or gallbladder outpouching to be seen.

The pancreas should be examined carefully, for Bower[5] lays stress on the frequency of associated pancreatic disease. The pancreas was mentioned in 12 cases, pancreatitis existing in 11 of these.

Case Report.—Hotel Dieu Hosp. Case No. G. 9207. Mrs. J. C., 41 years old, female. Admitted to hospital on 4-3-46, with a history that for 4 months she had had severe pain in right upper quadrant, requiring morphine for relief. The pain radiated to the right scapula and right shoulder. Associated with the pain, she had fever and chills, nausea and vomiting. For 2 months she had been jaundiced, on and off, with the

jaundice varying in intensity. She had lost 25 lbs. in weight since the beginning of her illness.

Her past history was essentially irrelevant.

Physical examination showed a middle aged, moderately obese woman who was markedly jaundiced. Pulse, temperature and respiration were within normal limits.

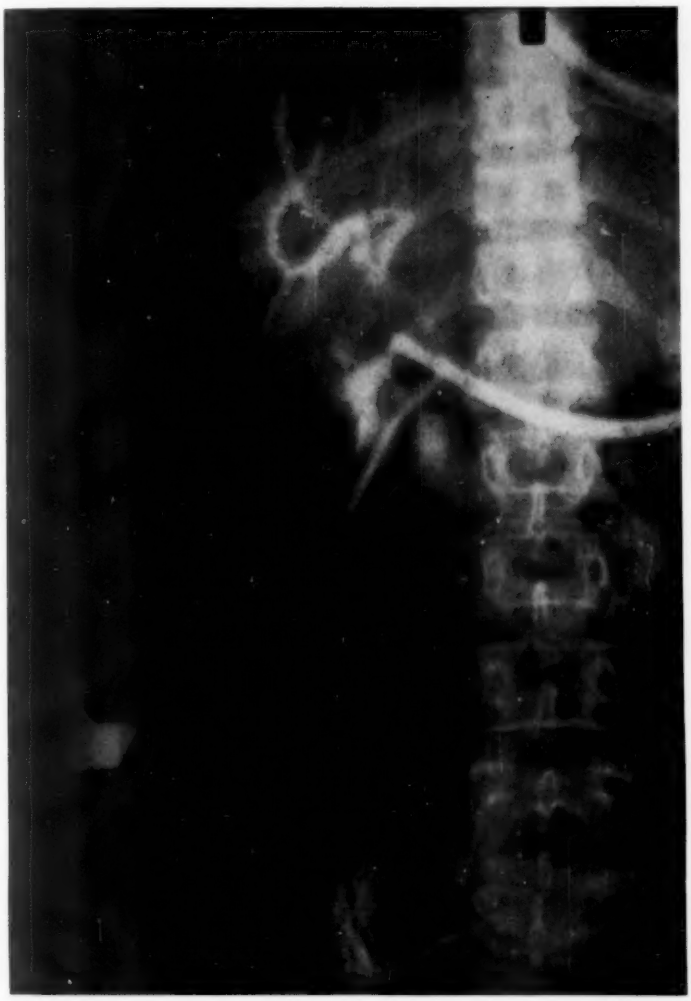

FIG. 2.—Cholangiogram, taken on operating table. All ducts are readily visualized but no vestige of gallbladder or cystic duct is seen.

Sclerae were yellow. Examination of the heart and lungs was normal. The abdomen was soft, with tenderness in right upper quadrant. There were no palpable masses. The liver was moderately enlarged, with edge two fingers breadth below costal margin. Spleen was not palpable.

Gallbladder visualization studies showed no gallbladder shadow and no stones were seen on the plain film. The radiologist made a diagnosis of gallbladder disease with

cystic duct obstruction. Her laboratory studies showed RBC 4,770,000 Hgb. 96.8 per cent WBC 8250 62 per cent polymorphonuclears. Negative Kahn and Eagle. Urine showed 2+ bile pigment and trace of albumin. Prothrombin time was 30 sec. (normal 15 sec.) All other laboratory studies were normal. A preoperative diagnosis of cholelithiasis, and cholecystitis with common duct stone was made. The patient was placed on the usual preoperative preparation which included vitamin K and when the prothrombin time was 15 sec. operation was carried out.

Operation was performed on April 6th, 1946, with nupercaine (1–1500) spinal anesthesia. The abdomen was opened through a right upper oblique incision. The undersurface of the liver was exposed and the region explored. No gall bladder was found. The outer border of the duodenum was found attached by a short wide fibrous band to the hilar region of the liver (Fig. 1). This was divided and the duodenum mobilized, exposing the common duct, which was about three times enlarged. No gallbladder fossa was seen. The common duct was explored upwards, to the point of junction with the two hepatic ducts. These were followed to their point of exit in the liver. No cystic duct, either fully developed or in the form of a stump was to be found. No cystic artery was seen. A large stone was palpated in the retroduodenal portion of the duct. The pancreas was hard and suggestive of pancreatitis. The common duct was incised, and the stone removed. A catheter was passed through the ampulla of Vater to demonstrate patency. The duct was irrigated with normal saline. A T-tube was placed in the duct and the duct closed with interrupted silk sutures. The appendix was removed. One Penrose drain was placed at the foramen of Winslow and brought out with T-tube through the stab wound. The wound was closed in layers with interrupted cotton sutures. With the patient still on the operating table, a cholangiogram was performed and both hepatic ducts, common duct and smaller intrahepatic ducts were well visualized (Fig. 2). No cystic duct or gallbladder was seen. The dye was seen passing readily through the duodenum into the upper jejunum.

Postoperative course was entirely uneventful. The patient was discharged on her 18th hospital day, with the jaundice rapidly receding and the T-tube still in place. The tube was removed on May 17th, 1946 and the stab wound rapidly healed. The patient has been seen at frequent intervals and she has remained entirely relieved of all her symptoms, with no further reappearance of her jaundice.

The possibility of an intrahepatic gallbladder was considered. It is felt that this possibility was eliminated, as surely as one can with a live patient, by the search at operation, the immediate postoperative cholangiogram which failed to show any cystic duct or gallbladder, and finally by the complete relief of the patient's symptoms during the period of over a year since her operation.

The possibility of an intrahepatic gallbladder must always be considered, when the gallbladder is not to be found in its usual position. There are a few of these cases reported, many of them containing calculi. One can with a fair degree of certainty, eliminate the possibility of this condition at the operating table by doing a cholangiogram, which should show all or part of the cystic duct and gallbladder, if they are present.

SUMMARY

A review of some of the findings in congenital absence of the gallbladder is presented. The necessity of considering the possibility of an intrahepatic gallbladder is brought out. The value of a cholangiogram at the operating table in eliminating this possibility is presented.

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HYDROCELE OF THE CANAL OF NUCK WITH LARGE CYSTIC RETROPERITONEAL EXTENSION

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TOWARD THE END of the eighteenth century Scarpa[1], in his book "Tumors of the Spermatic Cord," described a cystic tumor in the female inguinal canal which he referred to as "hydrocele of the canal of Nuck." Three prior instances had been reported by Aetius[2] 543 A.D., Plater[3] 1536 A.D., and Bertrondi[4] 1723 A.D. Moreover Desault 1737–1762, in the Journal d'Chirurg had described a case in detail in which the diagnosis was made certain by operation and excision of the sac. In 1832 an Italian surgeon, George Regnoli[5], Professor of Surgery at University of Pisa, prepared an exhaustive monograph on the subject, classified the various types and described an interesting case. Among others Chiari[6] recorded three cases in 1879, Wile[7] two in 1881, Coley[8] 14 in 1892, and Halstead and Clark[9] one in 1905. Halstead also referred to a case described by Thierhaber. In all Counsellor and Black[10] estimated that approximately 100 instances of hydrocele muliebris were reported from 1892 to 1939 and a total of not more than 350 cases have been reported until the present time.

Regnoli's description of hydrocele in the female still stands today with little alteration. He mentioned five types: (1) a diffuse hydrocele in cellular tissue enveloping the round ligament with transformation of the cellular tissue into a serous membrane, (2) an accumulation of fluid in a prolongation of peritoneum into the inguinal canal, the communication with the abdominal cavity remaining, (3) differs from the second only in the fact that the pouch of peritoneum no longer communicates with the abdominal cavity, (4) an encysted hydrocele in the connective tissue about the round ligament (similar to the first), (5) an accumulation of fluid in the remains of an old hernial sac.

When confined to the inguinal canal, hydrocele in the female first makes its appearance as a soft slightly tender often reducible mass in the inguinal region which is frequently mistaken for hernia. It may vary from the size of a hazel nut to that of a child's head. Seven of Counsellor and Black's cases were accompanied by hernia. Many were multilocular. Of 63 of his patients in whom the side of the lesion was known, Coley stated that the right side was involved in 36, the left in 25 and both sides in two. In many, but not all reported instances, the inguinal canal cyst communicated with the general peritoneal cavity.

Throughout the literature seven instances of hydrocele of the canal of Nuck with an intra-abdominal cystic extension have been discovered. In five

of the seven there were acute abdominal symptoms. One was diagnosed strangulated hernia and three intestinal obstruction.

In 1905 Halstead and Clark reported a case in a 42 year old colored woman who was hospitalized because of a tender swelling in the right inguinal region. An enlargement had been present in that area for 18 years but acute pain and tenderness for only eight days. She had vomited twice. On admission her temperature was 99°, white blood count 13,000. A preoperative diagnosis of strangulated hernia was made. At operation a cystic tumor containing an ounce and a half of fluid was found in the inguinal canal. This opened into a larger intra-abdominal cyst, which communicated with the general peritoneal cavity through a small opening.

Halstead and Clark also referred to a case of Thierhaber. A woman 42 years of age gradually developed a tumor in the inguinal region. This swelling suddenly increased in size and became painful to touch. At operation a mass the size of a goose egg was found which projected below the external ring into the labium majora. With a diagnosis of partially obstructed hernia, operation was performed and revealed a bilocular hydrocele one part within the abdominal cavity. The two chambers communicated but did not open into the free peritoneal cavity.

Three similar cases were reported by Chiari. In these there was inflammation of the wall of the sac which gave rise to symptoms of intestinal obstruction—vomiting, abdominal distention and obstipation. Operation revealed the nature of the condition.

Counsellor and Black reported 17 cases of hydrocele of the canal of Nuck. Among these one was a lemon sized, reducible, hour-glass type of tumor, an intra-abdominal part of which consisted of a large cyst in the iliac fossa. Another had a large cystic extension of the hour-glass type, chiefly intra-abdominal. There was no note of either of these hydroceles producing acute symptoms.

Because of the apparent rarity of this hour-glass type of hydrocele in the female, presentation of this additional case was felt to be justified.

CASE HISTORY

A 20-year-old colored girl, wife of an army corporal, was first seen in the hospital outpatient department on January 4, 1946, because of severe dysmenorrhea which she had had since puberty, manifested by abdominal cramps with each menstrual period for four years, and because of irregular vaginal bleeding of three years duration. A relatively normal menstrual period had occurred on November 5th, 1945, the last period having been on December 15, 1945. For about two years she had noticed a soft, non-tender swelling in the right inguinal region which caused no symptoms. Her past history had been non-contributory.

Physical examination revealed a fairly well developed girl in no apparent discomfort. Heart, lungs, pharynx, bones and joints were normal. There was a soft, slightly tender, reducible mass half the size of a walnut in the right inguinal region. The left inguinal region was normal. On pelvic examination cervix and perineum were normal. The uterus was moderately enlarged, firm, retroverted and tender. No masses were felt in the adnexia but there was tenderness in both vaults.

The urine was straw colored, hazy, contained no albumin or sugar but did reveal a few epithelial cells and 1–3 white cells per H.P.F. Hematocrit 39. Kahn negative.

With a diagnosis of right inguinal hernia and mild pelvic inflammatory disease she was admitted to the hospital on January 12th, 1946. Hospital history, physical examination and laboratory studies confirmed those recorded by the outpatient department, and operation for right inguinal hernia was scheduled for January 18, 1946.

Operation.—Under satisfactory procaine and pontocaine spinal anesthesia a right inguinal incision was made from a point one inch medial to the anterior superior spine downward to the pubic tubercle. This incision was carried down to the aponeurosis of

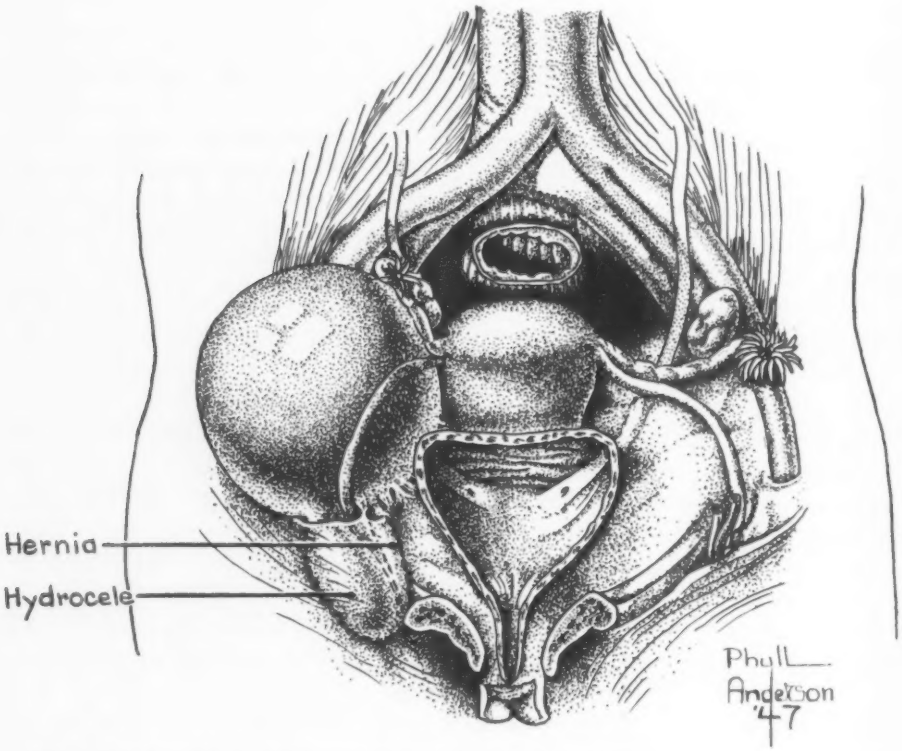

FIG. 1.—Hydrocele of the canal of nuck with retroperitoneal cystic extension and accompanying hernia.

the external oblique muscle which was split in the direction of its fibers. The round ligament was gently dissected free from the surrounding tissues. Along the lower part of the ligament there were a number of loculated pockets containing clear yellow cystic fluid. More laterally these seemed to unite to form a cystic cavity, sausage shaped, containing about 10 cc. of clear fluid. At the internal ring this cystic space was found to be continuous with a large intra-abdominal cyst slightly larger than an orange. Connecting the two cystic spaces was a somewhat narrower neck. By slipping a finger through this neck the distant walls of the intra-abdominal cyst could be felt. On exploring further along the round ligament a true indirect inguinal hernia which communicated with the general peritoneal cavity was discovered. The round ligament lay between the cyst and hernia.

Because of the possibility that the cyst might be a diverticulum of the bladder, the bladder was catheterized on the operating table. It contained the usual amount of urine and did not communicate with the cyst. To explore the lesion more thoroughly the incision was then extended across the midline, the fascia dissected upwards, the rectus muscles separated vertically and the peritoneal cavity opened. It was then discovered that the cyst was entirely retroperitoneal accompanying the round ligament, its fundus lying between the layers of the broad ligament. Gradually from the inguinal and intra-peritoneal approaches the cyst was dissected free and wall delivered into the inguinal incision lateral to the hernia. The hernial sac was dissected out and tied off, the lower fanned-out portion of the round ligament excised and a Bassini type of repair of the inguinal canal performed.

The postoperative convalescence was entirely without incident.

Examination on July 13, 1946 revealed an enlarged retroverted uterus, slight tenderness of both adnexia and a well healed inguinal scar. Except for some continued irregularity of her menstrual periods she was symptom free.

In the case noted above, as in the two reported by Counsellor and Black the cyst did not cause symptoms, merely the presence of a mass in the inguinal region. The patients of Halstead and Clark, Thierhaber and Chiari however were treated because of acute episodes suggesting intestinal obstruction or strangulated hernia. In at least one of Chiari's cases there was evidence of acute inflammation of the wall of the cyst. On this account it seems reasonable to suppose that the acute symptoms described by these authors were due to inflammation of the adjacent peritoneum.

SUMMARY

A review of the literature of hydrocele of the canal of Nuck is presented. Emphasis is placed on seven previously reported cases of an hour-glass type in which part of the hydrocele was intra-abdominal. Another case of this type is described together with a suggested method of treatment.

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LIPOMA OF THE DUODENUM CAUSING MELENA

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Benign tumors of the duodenum are rare. Silent hemorrhage is often the only symptom of tumors of the small intestine. This paper is the report of an interesting case of an ulcerated lipoma of the duodenum, the only symptom being intermittent melena.

REPORT OF CASE

History and Physical Examination.—C. K., a 70-year-old white male, was referred to the George F. Geisinger Memorial Hospital for gastro-intestinal studies because of a history of intermittent tarry stools for two years. The patient had enjoyed excellent health until early October 1944, when he had a sudden attack of weakness and dizziness. Following this he observed that his stools were tarry black in color for one week. There was no abdominal pain, nausea, or vomiting associated with the attack. No history of intolerance for fatty foods could be elicited. Similar, milder episodes had occurred subsequently at irregular intervals during the past two years. These were never associated with any abdominal symptoms. Between attacks the patient had no complaints. There was no weight loss. The last attack was one month before admission.

Physical examination revealed a well developed, rather obese white male in no distress. Weight was 195 pounds. Temperature, pulse, respiration were normal. BP 140/78. There were no masses or tenderness in an obese abdomen. The remaining portion of the examination was essentially negative.

Laboratory Examination.—Examination of the urine was negative. Flocculation reaction for syphilis was negative. Blood studies revealed a mild hypochromic anemia with erythrocytes 3,950,000, leukocytes 2,450, hemoglobin 62 per cent, and color index 0.8. Tests for occult blood in the stools were negative.

Roentgenologic Examination.—Roentgenologic examination of the upper gastro-intestinal tract revealed no evidence of organic disease of the esophagus or stomach. There was no gastric residue from the motor meal ingested six hours previously. The duodenal bulb was normal in size and contour. In the third portion of the duodenum there was an elongated mass 5.5 by 3 centimeters in size. No ulceration of the surface of this mass could be detected. There was no obstruction proximal to the mass which almost filled the lumen. Because of the position of the lesion and the habitus of the patient no contributory pressure films could be obtained. Films taken subsequent to fluoroscopy showed an apparently normal small intestine distal to the ligament of Treitz. The roentgenologic impression was "benign tumor of the duodenum possibly a leiomyoma."

Surgical Findings.—A laparotomy was performed on June 26, 1946, by Dr. Harold L. Foss. Following exposure of the second and third portions of the duodenum, the tumor was readily palpated within the lumen. On opening the duodenum a soft pedunculated polypoid mass approximately 5 cm. long was found. The base of the pedicle was ligated and the tumor removed. Postoperative course was uneventful. On a follow-up visit several months subsequent to operation the patient stated there has been no recurrence of bleeding.

Pathologic Findings.—Pathologic examination of the specimen showed a soft ovoid mass measuring 5 by 3.5 by 2 cm. in size. The surface was smooth and glistening and had the appearance of normal mucosa. At the distal pole there was a small bluish scar with tiny petechial hemorrhages about it. The cut pedicle measured 1.0 cm. Through the cut surface of the pedicle a soft yellow inner substance could be seen. Section revealed a normal reflection of mucosa over the tumor. The central mass was composed of three lobules of soft yellow adipose-appearing tissue.

Microscopic section confirmed the gross impression of adipose tissue. The mucosa was normal. Diagnosis was lipoma.

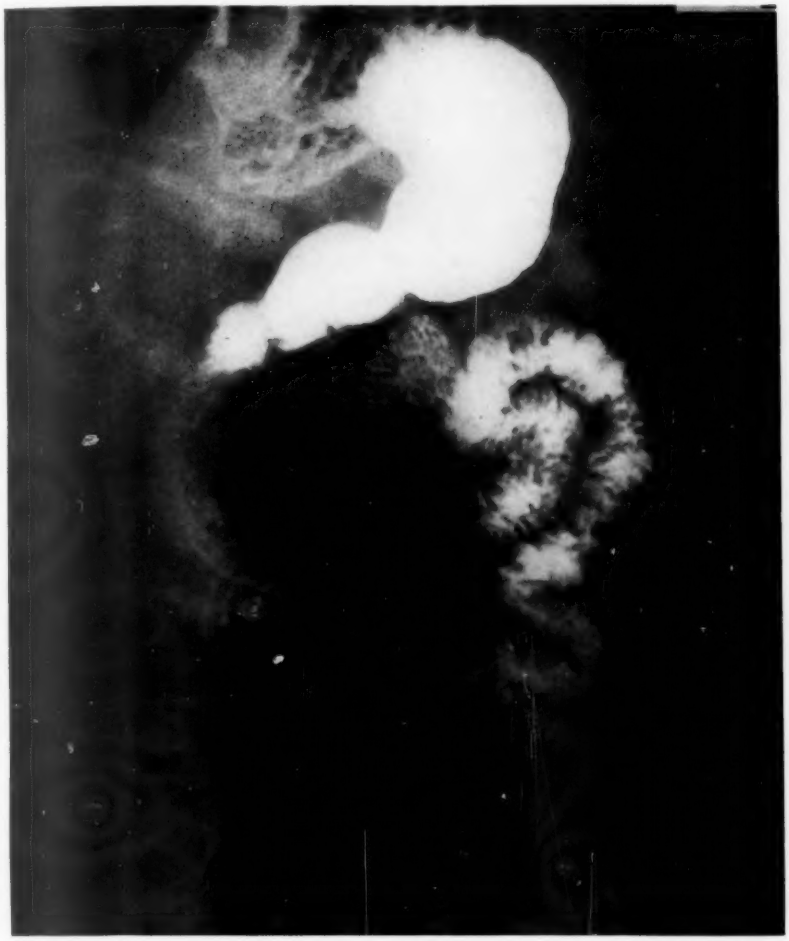

FIG. 1.—Roentgenogram showing tumor of duodenum.

COMMENT

The incidence of benign tumors of the small intestine is given by Raiford[1] in his extensive survey as approximately 4 per cent of all gastro-intestinal tumors. Lipomas form only a small percentage of these. Generally these neoplasms are single but infrequently are multiple. The size varies from that of a pea to more than a man's fist. The common form is polypoid although a fair number of the sessile variety are reported.

There have been less than 20 lipomas of the duodenum reported to date. Most of these were incidental findings at autopsy. Degner's case as reported in Comfort's[2] comprehensive survey of submucous lipomas is the only proven case with symptoms we can discover.

Although benign tumors are rare, when they occur they often produce serious symptoms. In cases of adult intussusception of the small bowel, lipomas have been traced as the etiologic factor in 44 per cent—Schottenfeld.[3]

Good and MacCarty[4] list three primary clinical manifestations of benign tumors of the intestine. They are: (1) loss of blood as by hematemesis or melena, (2) presence of a mass, (3) evidence of obstruction.

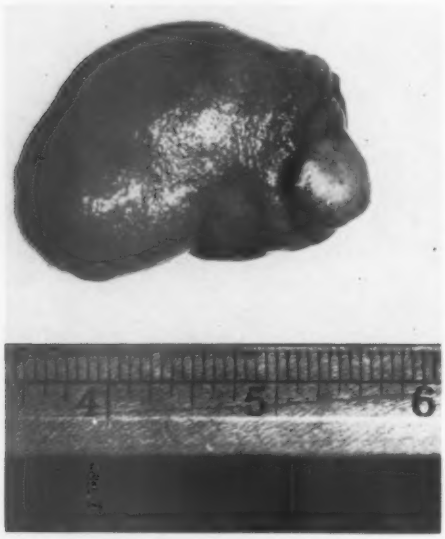

FIG. 2.—Photograph of gross specimen.

Intussusception in the duodenum is very unusual as it is fixed for the most part. Also, as the duodenum is deeply situated in the abdomen, palpation of a soft mass would be difficult or impossible. Hemorrhage follows ulceration of the stretched atrophic mucosa. There have been ten cases of spontaneous passage of lipoid tumors with bloody stools, presumably subsequent to avulsion of these polypoid growths.

It is obvious that if the diagnosis of benign small intestinal tumor is to be made prior to operation or necropsy, it must be made by the roentgenologist. Most tumors of the upper gastro-intestinal tract can be discovered in the course of an ordinary roentgenoscopic study of the esophagus, stomach and duodenum. Tumors distal to the ligament of Treitz require special small intestinal studies such as are described by Golden[5] or Schatzki.[6]

It is not always possible to make a roentgenologic differentiation between benign and malignant tumors. There is a high incidence of serious complications in all small intestinal tumors. For these reasons we believe that when the diagnosis of small intestinal tumor is made, the patient should be operated upon even in the absence of symptoms.

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A NEW METHOD OF RESTORING CONTINUITY OF THE ALIMENTARY CANAL IN CASES OF CONGENITAL ATRESIA OF THE ESOPHAGUS WITH TRACHEO-ESOPHAGEAL FISTULA NOT TREATED BY IMMEDIATE PRIMARY ANASTOMOSIS

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DECISION CONCERNING the treatment of congenital atresia of the esophagus with tracheo-esophageal fistula in any given case requires that the surgeon establish a general policy of management of this anomaly. At first this condition was regarded as hopeless. It was finally shown by Ladd[1] and others that the lives of some of these unfortunate infants could be saved by means of a multiple stage procedure consisting of closure of the fistula, the establishment of a cervical esophagostomy, and the creation of a gastrostomy for feeding purposes. In a few of these patients it has been possible with much effort to construct an external esophagus using tubed flaps of skin, segments of jejunum, and so forth. This procedure has admittedly been attended by many difficulties and disappointments and few real successes. More recently it has been shown conclusively by Haight,[2] Ladd and Swenson,[3] and others that in many cases it is possible to close the fistula and to perform a primary anastomosis so as to create an intact esophagus. Perfections in technic resulting from increased experience have made it possible to perform this operation with a relatively high percentage of successful results in suitable cases. It is obvious, therefore, that if enough length of esophagus is available, closure of the fistula followed by the performance of a primary anastomosis is the operation of choice.

There remains, however, the problem of how to handle the case where it is impossible to carry out this ideal procedure. Attempts to construct an external esophagus in these cases have been so discouraging that some surgeons have expressed the opinion that it might be better to allow such unfortunate infants to die rather than to preserve them only for a life of suffering or semi-invalidism. No doubt the majority of surgeons would feel obliged to prolong the infant's life if possible by closing the fistula and performing a cervical esophagostomy and a gastrostomy. The parents of the child, on the other hand, sometimes adopt the other point of view: that it is better to allow the child to die, hardly having lived, than to allow him to live with the handicap which is inevitable for him. This opinion must be regarded with sympathy.

It is apparent, therefore, that if a method of restoring the continuity of the alimentary canal without having recourse to the multiple stage external esophagoplasty usually advocated could be developed, it would make it easier for surgeons and parents alike to decide in favor of an attempt to preserve the life of the child in cases where a primary anastomosis cannot be made. A method to accomplish this end by performing an intracervical

esophagogastric anastomosis after pulling the stomach up through the chest is herein set forth.

NATURE OF THE PROBLEM

The principal obstacles to be overcome are three in number:

(1) *The long distance between the short proximal segment of esophagus and the fundus of the stomach.* That the stomach can be mobilized sufficiently to place the fundus in the apex of the left pleural cavity has already been demonstrated.[4] In the cases of congenital atresia which have been treated by cervical esophagostomy and gastrostomy, the anastomosis must be made several centimeters above the level of the clavicle because of the invariably short proximal stump of the esophagus which must be used. If the mobilization of the stomach is complete, however, sufficient length can be obtained to bring the fundus as high in the neck as required. This means, in addition to the usual freeing of the fundus and division of the left gastric and left gastro-epiploic vessels, the complete division of the gastrohepatic and gastrocolic ligaments as far as the level of the pylorus. This dissection must be done with great care to avoid injury to the right gastric and right gastro-epiploic vessels and their anastomotic arches along the lesser and greater curvatures of the stomach.

(2) *The presence of a gastrostomy opening.* The performance of a gastrostomy is, of course, necessary in the type of congenital atresia which does not lend itself to a primary anastomosis. But the presence of such a fistula does not present a very serious handicap because it can be closed readily after the stomach has been freed from its attachment to the anterior wall of the abdomen. It is important, however, to close the opening in the gastric wall in such a way that no shortening of the stomach will result. This is accomplished by placing the sutures across the stomach so that when they are tied, the opening is infolded in the direction of its long axis. This produces slight narrowing of the lumen which is of no consequence and lengthwise shortening is avoided.

(3) *The passage of the fundus of the stomach from the thoracic cavity into the neck.* This presents a somewhat difficult problem which can best be solved by performing what may seem at first to be a rather radical maneuver. It is not possible to develop by dissection a large enough passageway from the superior mediastinum into the base of the neck to make room for the relatively large fundus of the stomach. Furthermore, if the fundus is brought out through a short anterior intercostal incision and then up into the neck through a subcutaneous tunnel, the pressure of the overlying skin and fascia would cause too much compression of the stomach and anastomosis against the underlying structures. Ample room for the fundus can be obtained, however, by resecting the inner one-half of the clavicle and a comparable segment of the first rib so that the fundus can be passed into the neck without pressure or constriction to meet the high-lying proximal esophageal segment.

DESCRIPTION OF THE OPERATION

The operation is performed in one stage as follows:

(1) *First Step of the Procedure—Mobilization of the Stomach and Resection of the Distal Segment of Esophagus.* The patient is placed on his right side with the left arm drawn forward out of the way. A long intercostal incision extending the entire length of the eighth interspace is made. A small size rib spreader is inserted. The diaphragm is incised from a point close to its costal insertion through the margin of the esophageal hiatus. The rudimentary distal segment of the esophagus is freed through an incision in the mediastinal pleura and removed by severing it just above the cardia. The remaining stump is inverted with a purse-string suture of silk reinforced by several Lembert sutures of the same material. The anterior wall of the stomach is then freed from its attachment to the abdominal wall and the gastrostomy opening is closed with two layers of fine silk sutures. The attachments of the fundus of the stomach are divided, including the gastrolienal ligament with its enclosed vasa brevia which must be ligated. The left gastro-epiploic vessels are tied and cut and the entire gastrocolic ligament is incised all the way to the level of the pylorus, taking care to avoid injury to the arcade of vessels along the greater curvature of the stomach which is supplied by the right gastro-epiploic vessels. The left gastric artery and vein are tied and cut. The gastrohepatic ligament is then incised as far as the level of the pylorus. Here likewise the integrity of the vascular arches along the lesser curvature, which are supplied by the right gastric vessels, must be preserved. It is important to mention in this connection that the left gastric artery should be tied and cut close to its origin from the celiac axis so as to preserve the peripheral branches which form the greater portion of the arcade along the lesser curvature. The stomach is now sufficiently well mobilized to allow the fundus to be pulled up through the thorax to the base of the neck or above.

A strand of silk is then passed with a fine needle through the wall of the fundus of the stomach, which has been pulled through the diaphragm and up behind the hilum of the lung. A trochar-pointed needle (Keith) is substituted for the fine needle on the end of the silk strand. This needle is thrust from within through the first left intercostal space anteriorly and by means of traction on the attached thread of silk, the fundus of the stomach is held up against the anterior thoracic wall during the closure of the thoracotomy incision until it is needed for the performance of the anastomosis in the neck (Fig. 1, No. 4).

To complete the first stage of the operation the lung is expanded by the anesthetist and the thoracotomy incision is closed, using pericostal sutures of fine chromic catgut to approximate the ribs and interrupted sutures of fine silk in the remaining layers.

(2) *Second Step of the Procedure—Performance of the Intracervical Esophagogastric Anastomosis.* After the closure of the thoracotomy incision

has been completed, the patient is turned on his back and a vertical incision is made from the esophagostomy stoma above to the level of the second rib below. The pectoralis major muscle is incised close to its attachment to the sternum and reflected laterally, at the same time separating its insertion to the medial portion of the clavicle. The sternal and medial clavicular insertions of the sternocleidomastoid muscle are severed and re-

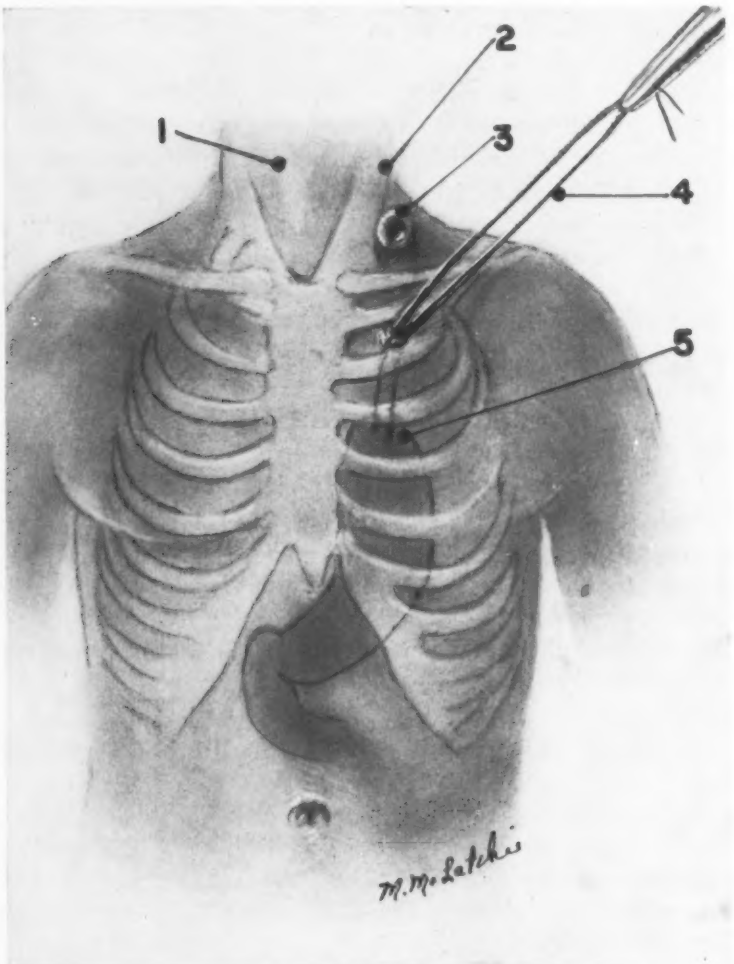

FIG. 1—Diagram illustrating the method of maintaining the position of the fundus of the stomach in the apex of the chest during closure of the thoracotomy incision. (1) Thyroid gland (2) Sternocleidomastoid muscle (3) Cervical esophagostomy stoma which is to be used for the anastomosis (4) Silk thread which is brought out through the first intercostal space and used to hold the fundus of the stomach in the apex of the chest until the thoracotomy incision has been closed. After the cervical incision has been made and the left pleural cavity opened from above, the stomach is drawn up into the neck as shown in Figure 2. *Note:* No attempt is made in this drawing to illustrate the relations of the stomach to the heart and left lung.

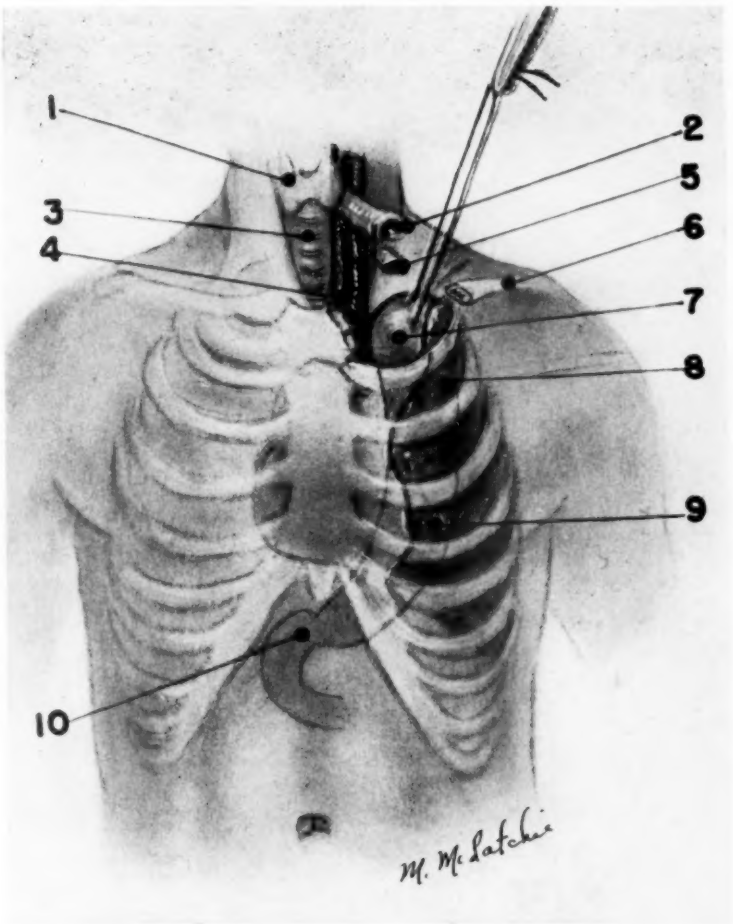

FIG. 2—Diagram illustrating the method of bringing the stomach up through the left thoracic cavity and into the base of the neck. For the sake of clearness, the left sternocleidomastoid and pectoral muscles have been omitted from the drawing instead of being shown retracted laterally as is the case in the operation. The stomach lies behind the heart and the hilum of the left lung. The fundus of the stomach is shown being drawn up out of the pleural cavity into the neck. As this is done it passes medially to the apex of the lung and in front of the subclavian artery and vein. The short proximal length of esophagus passes anterior to the carotid sheath. The anastomosis therefore lies in front of the carotid vessels. (1) Thyroid gland (2) Stoma and short proximal end of the esophagus (3) Trachea (4) Carotid vessels (5) Cut end of the first rib after resection of the anterior segment (6) Lateral one-half of the clavicle showing cut end after resecting the medial one-half (7) Fundus of the stomach with temporary silk thread still in place (8) Left lung lying anterior and lateral to the stomach (9) Apex of the heart (10) Duodenum pulled somewhat to the left.

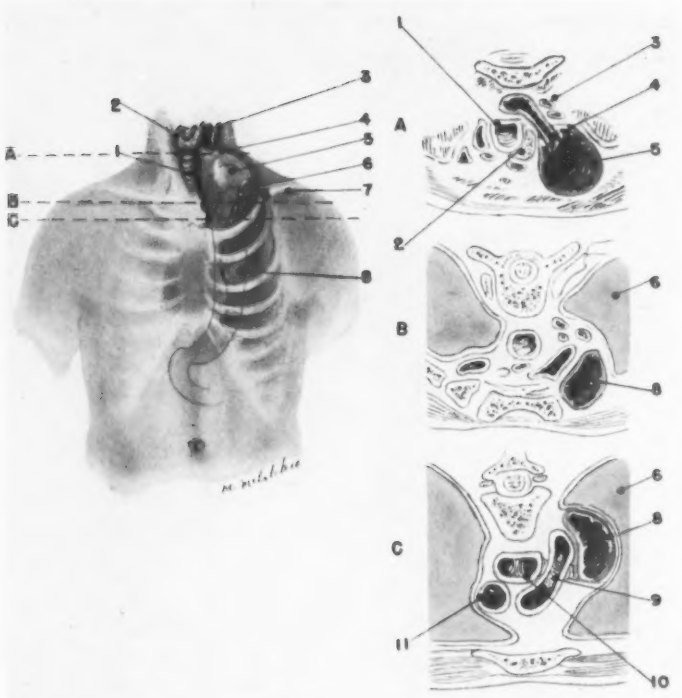

FIG. 3—Diagram showing the relations of the stomach after the completion of the anastomosis (1) Trachea (2) Thyroid gland (3) Carotid vessels (4) Esophagogastric anastomosis (5) Fundus of the stomach (6) Apex of the left lung (7) Lateral half of the clavicle (8) Level of the body of the stomach where it lies behind the hilum of the lung.
Cross Section A Level of the esophagogastric anatomosis in the neck (1) Trachea (2) Thyroid gland (left lobe) (3) Carotid and jugular vessels (4) Esophagogastric anastomosis (5) Fundus of the stomach.
Cross Section B Level of the dome of the pleural cavity (6) Left lung (8) Stomach.
Cross Section C Level of aortic arch (6) Left lung (8) Stomach lying behind the hilum of the left lung (9) Aortic arch (10) Trachea looking down at the bifurcation (11) Superior vena cava.

tracted laterally. The incision is then carried around the esophageal stoma and enough of the end of the esophagus is freed to make it possible to perform an anastomosis. This mobilization of the esophageal segment must not be too extensive for fear of jeopardizing the blood supply to the end which must be preserved for the anastomosis.

The medial half of the clavicle and a corresponding segment of the left first rib and costal cartilage are resected extraperiosteally. This produces a large opening from the base of the neck behind the lower end of the sternomastoid muscle into the apex of the left pleural cavity through which the

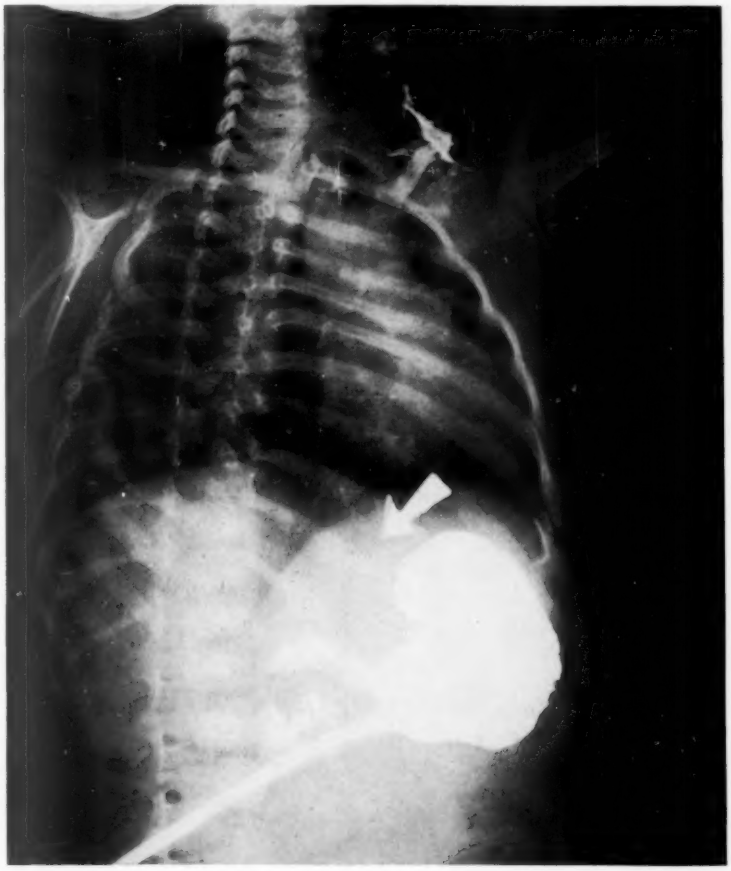

FIG. 4—Case W. C. Pre-operative roentgenogram showing barium taken orally after spurting onto the neck through the esophageal stoma, and barium put into the stomach through the gastrostomy catheter with a faint trickle of barium extending several centimeters into the distal blind segment of the esophagus. (Arrow)

fundus of the stomach can be drawn easily and without danger of compression. The temporary fixation of the fundus of the stomach is eliminated by cutting the silk suture which was used to attach it to the tissues of the first intercostal space. The fundus is pulled up behind the apex of the lung into the lower portion of the neck (Fig. 2). A short incision is made in the posterior wall of the fundus close to its apex and an anastomosis consisting of three layers of interrupted fine silk sutures is made. Careful approximation of mucosa to mucosa and muscle-edge to muscle-edge constitute the inner and middle layers. The outer layer is of interrupted mattress sutures. Several interruped sutures are used to fix the fundus to the tissues surrounding the region of the anastomosis. The method of pulling up the fundus of the stomach and the location of the esophagogastric anastomosis

at a high level in the neck is illustrated in Figures 2 and 3. The wound is closed by re-suturing the lower end of the sternocleidomastoid muscle and the cut edge of the pectoral muscle to the sternum and placing a layer of fine silk sutures in the subcutaneous fat and another in the skin. No drainage is used.

(3) *Third Step of the Procedure—Closure of the Abdominal Wall Portion of the Gastrostomy.* The edges of the former gastrostomy incision are excised. The fascia and peritoneum are identified, and a layer by layer closure of the small opening in the abdominal wall is brought about using interrupted fine chromic catgut sutures, with silk to the skin.

POSTOPERATIVE CARE

The patient's condition must be maintained during the first week or ten days after operation by means of the intravenous administration of solutions containing the necessary electrolytes, glucose, amino acids, and vitamins. Fluids may be allowed by mouth after five to seven days. By the end of 12 to 14 days the child should be able to take a diet suitable for his age.

Penicillin in doses depending upon the age of the child is administered until the danger of postoperative sepsis and pulmonary complications is over (approximately one week). The child should be kept in an oxygen tent during the first few days to ease the burden on the respiratory mechanism.

CASE REPORT

As an illustration of the utilization of this method of restoring continuity of the alimentary tract after the performance of a cervical esophagostomy and a gastrostomy, the following case report is submitted:

W. C., a white male infant, age 21 months, was admitted to the Baker Memorial unit of the Massachusetts General Hospital on June 19 1947, referred by Dr. J. C. McCann of Worcester, Massachusetts. The history was as follows:

On the third day following birth it was discovered that the patient had a congenital tracheo-esophageal fistula. He was taken immediately to a children's hospital where he was operated upon. Through a right thoracotomy incision the fistula was closed and later a cervical esophagostomy and a gastrostomy were performed. After a stormy convalescence complicated by the occurrence of bilateral pneumonia and dehiscence of the gastrostomy wound requiring secondary suture, the patient recovered and since then had developed at a normal rate. Although given to understand at first that a connection between the esophagus and the stomach would be made, the parents were finally told that it was not worth while attempting any further surgery. This decision was very disturbing to them because the child was eager to eat and swallow things and it was most pathetic to watch him when everything he took by mouth came out through the skin of his neck. They were anxious to undertake any risk whatever, no matter how great, to make it possible for the child to eat in a normal fashion.

On examination at the time of admission to the Baker Memorial the child was a healthy appearing boy for his age. He walked normally but did not talk very much. A large catheter led into the stomach through a rather irritated-looking stoma which was in the center of a longitudinal abdominal incision through the left rectus muscle. In the left side of the neck about 2 cm. above the clavicle there was a cervical esophagostomy. The union of the esophageal mucosa to the skin was smooth. It was necessary to keep a dressing on the patient's neck at all times.

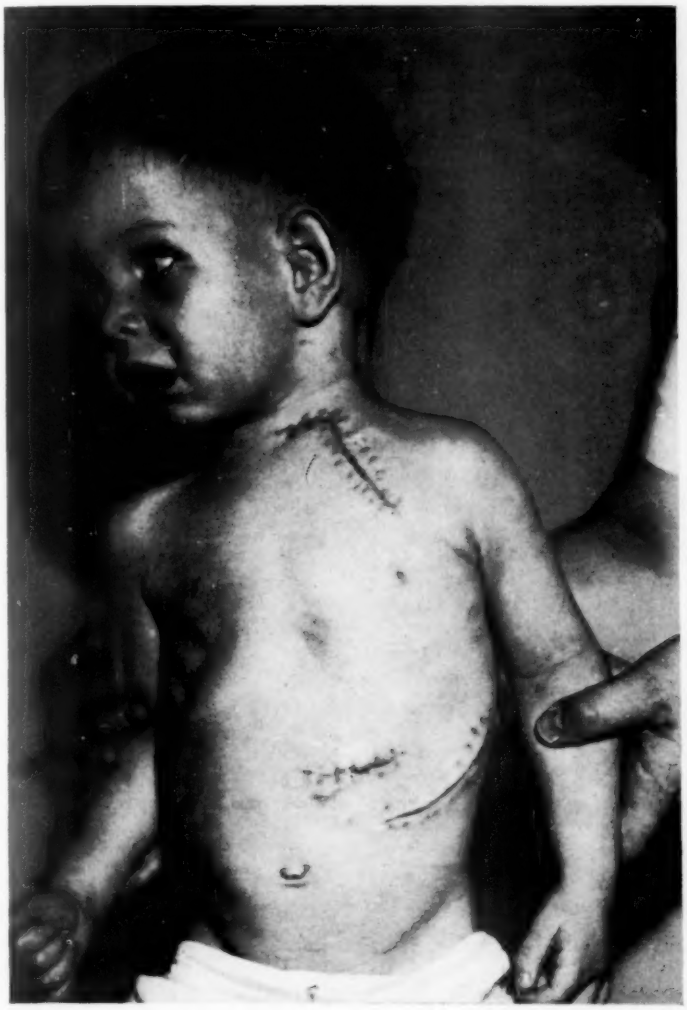

FIG. 5—Case W. C. Photograph taken three weeks after operation showing the closed gastrostomy incision in the anterior abdominal wall, the cervical incisional scar, and the anterior portion of the thoracotomy incisional scar.

Barium given by mouth passed immediately into the dressing at the left side of the neck. Barium introduced through the gastrostomy tube did not pass up into the esophagus sufficiently far to demonstrate with certainty the lower segment, but there appeared to be approximately 6 cm. of esophagus. Barium passed readily through the pylorus into the duodenum (Fig. 4).

Laboratory examinations showed the urine to be light amber in color with an acid reaction and a specific gravity of 1.030. There was no albumin, sugar, diacetic acid, or bile. The sediment contained 4 white blood cells, 2 epithelial cells, and mucin. The white blood count was 8600, the photo. hemoglobin 12.2 Gm. There were 57 polymorphonuclear leucocytes, 37 small lymphocytes, and 6 monocytes. The stained spe-

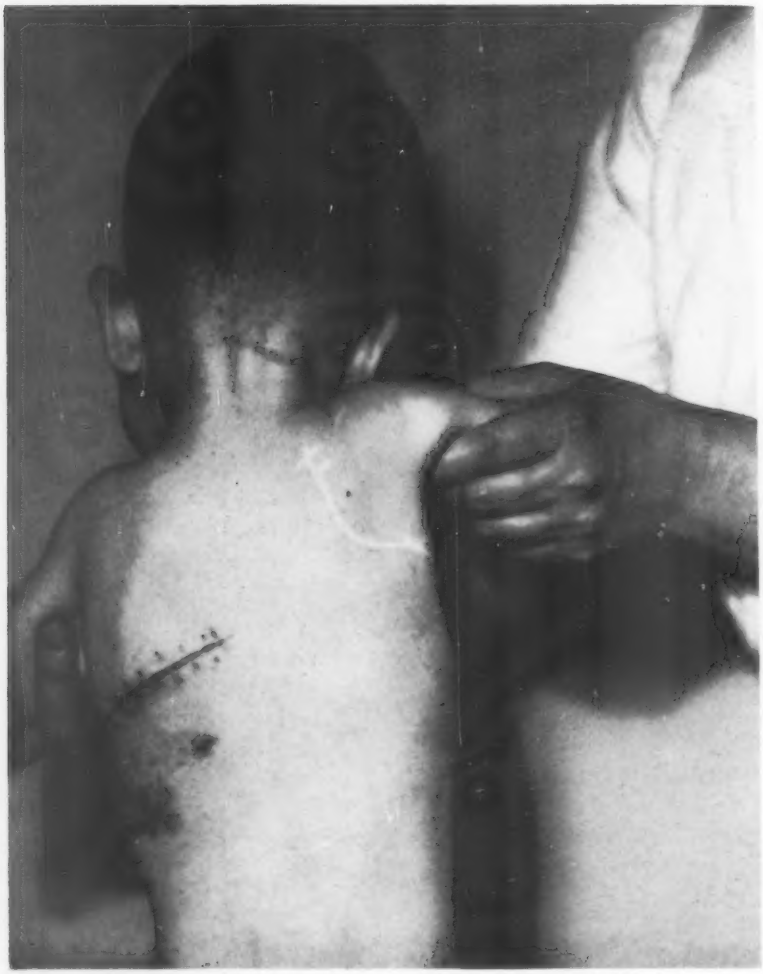

FIG. 6—Case W. C. Photograph taken three weeks after operation showing posterior portion of the thoracotomy incisional scar. The scar of the previous thoracotomy on the right side shows in this view.

cimen showed a few polymorphonuclear leucocytes, moderate achromia and variation in size, and a rare stippled red blood cell. The platelets appeared fairly normal. The plasma protein was 6.7 Gm. per cent. The prothrombin time was 17 seconds.

After ten days of observation and preparation for operation a transthoracic and trans-cervical partial esophagectomy with esophagogastric anastomosis was performed according to the technic already described. Postoperatively hydration and nutrition were maintained by continuous intravenous drip. This was accomplished with difficulty because of the small size of his veins and the fact that many of them had been used before. He took practically nothing by mouth until his fourteenth postoperative day. He then began to take food in small amounts but not sufficient for nourishment, so that a Levine tube was passed through the anastomosis into his stomach and he was fed by gavage until about the 20th day after operation when he began to eat normally. He lost about 4 pounds while he was in the hospital. After his mother had been instructed

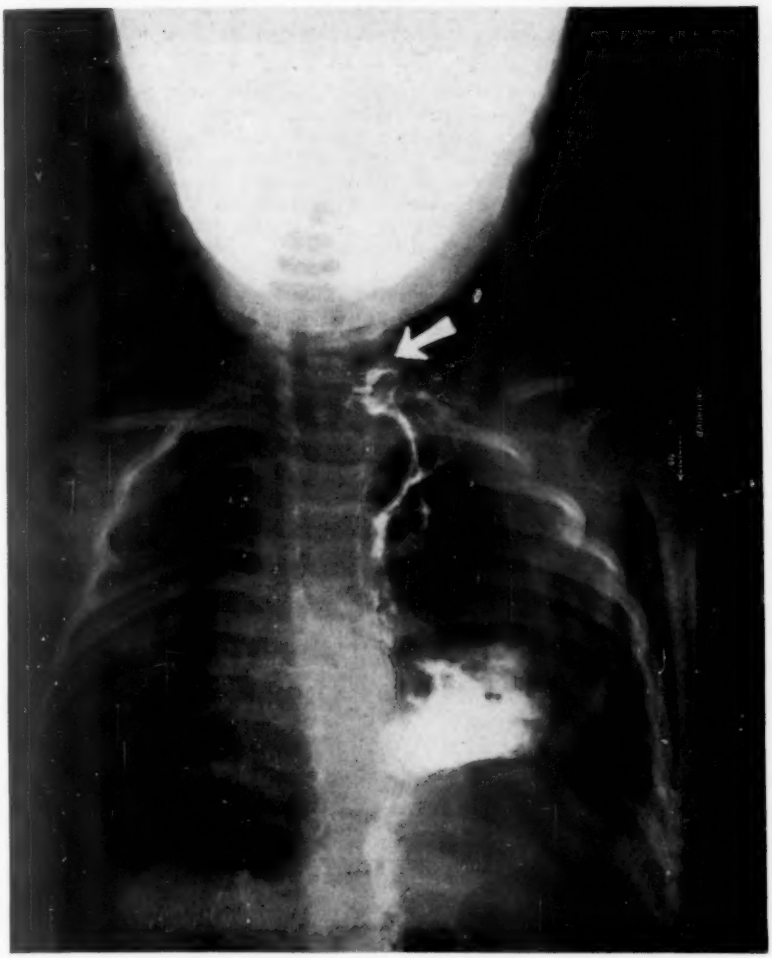

FIG. 7—Case W. C. Postoperative roentgenogram after ingestion of barium. The anastomosis is indicated by the arrow. Just below are the rugal folds of the fundus of the stomach. The remainder of the stomach as it lies in the left pleural cavity behind the hilum of the lung contains air and a small amount of barium. The cut ends of the first rib and clavicle are easily seen. The lung lies around and to the side of the stomach.

in his feeding and he was ready to go home, he had a gastro-intestinal upset which delayed his discharge from the hospital for about a week.

When the patient was brought to the office for a postoperative examination three weeks after leaving the hospital, he looked very well and, according to his mother, was eating a liberal diet in large quantities. The incisions were well healed. There was some ballooning out of the soft tissues at the base of the left side of the neck during inspiration. His weight was about the same as when he left the hospital, but he had apparently adjusted completely to conditions at home and was making steady progress. A more recent report from his physician states that the child is eating a diet normal

for his age. The amount of food consumed is normal, but his weight gain has been slow. His stools are normal and there is no obvious evidence of physiologic disturbances resulting from the displacement of his stomach through his chest into his neck.

ADDENDUM.—Since this operation was performed, the same technic has been used for a subtotal esophagectomy and intracervical esophagogastric anastomosis in a case of carcinoma of the esophagus located in the region behind the manubrium of the sternum. No satisfactory procedure has been available previously for use in such cases. A growth at that level is too low for the Wookey operation,[5] which can be applied only in cases where the tumor is in the cervical segment, and too high for the application of the operation of transthoracic esophagectomy with a high intrathoracic esophagogastric anastomosis.[6] The patient, a man 56 years of age, has made an uneventful recovery and is eating in a normal fashion.

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LETTER TO THE EDITOR

Dear Sir:

Dr. D. B. Phemister calls my attention to a bibliographic error in the December 1947 article by myself and coworkers on Hemorrhagic Shock. **Reference #16 should read:**

Phemister, D. B. and C. H. Laestar: Local Fluid Loss, Nerve Stimuli and Toxins in the Causation of Shock. Ann. Surg., **121:** 803, 1945.

Phemister, D. B.: The Mechanism and Management of Surgical Shock. J.A.M.A. **127:** 1109, 1945.

Parsons, E. and D. B. Phemister: Hemorrhage and "Shock" in Traumatized Limbs. Surg., Gynec. and Obst., **51:** 196, 1930.

Reference #7 should have in addition to the one given the following:

Phemister, D. B., C. H. Laestar, L. Eichelberger and R. J. Schachter: Afferent Vasodepressor Nerve Impulses as a Cause of Shock. Ann. Surg., **119:** 26, 1944.

JACOB FINE, M. D.